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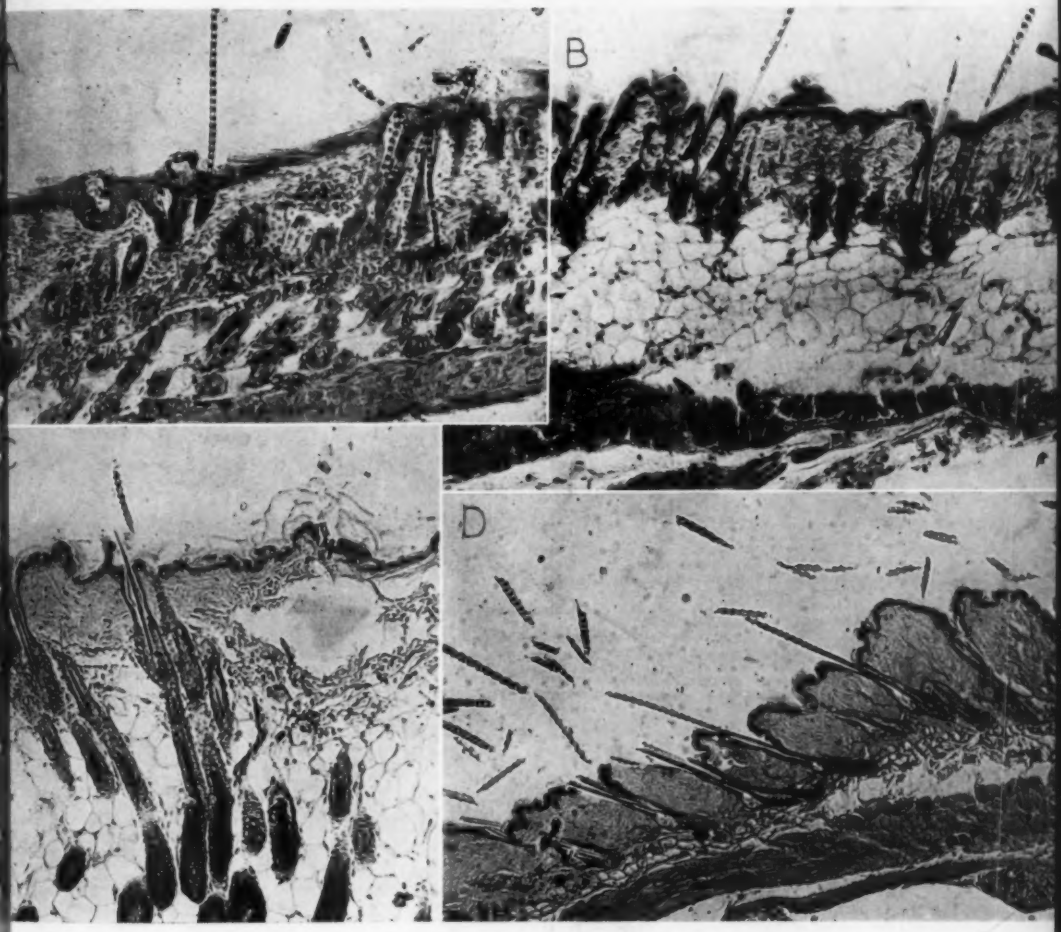

CLOSE-UP OF MOUSE NUDISM AND NORMALITY

(SEE PAGE 213)

Silky Plumage in the Pigeon

X-Ray Induced Chromosome Rearrangements

A Dominant Crippling Defect of the Extremities

VOLUME 30 MAY, 1939 NUMBER 5

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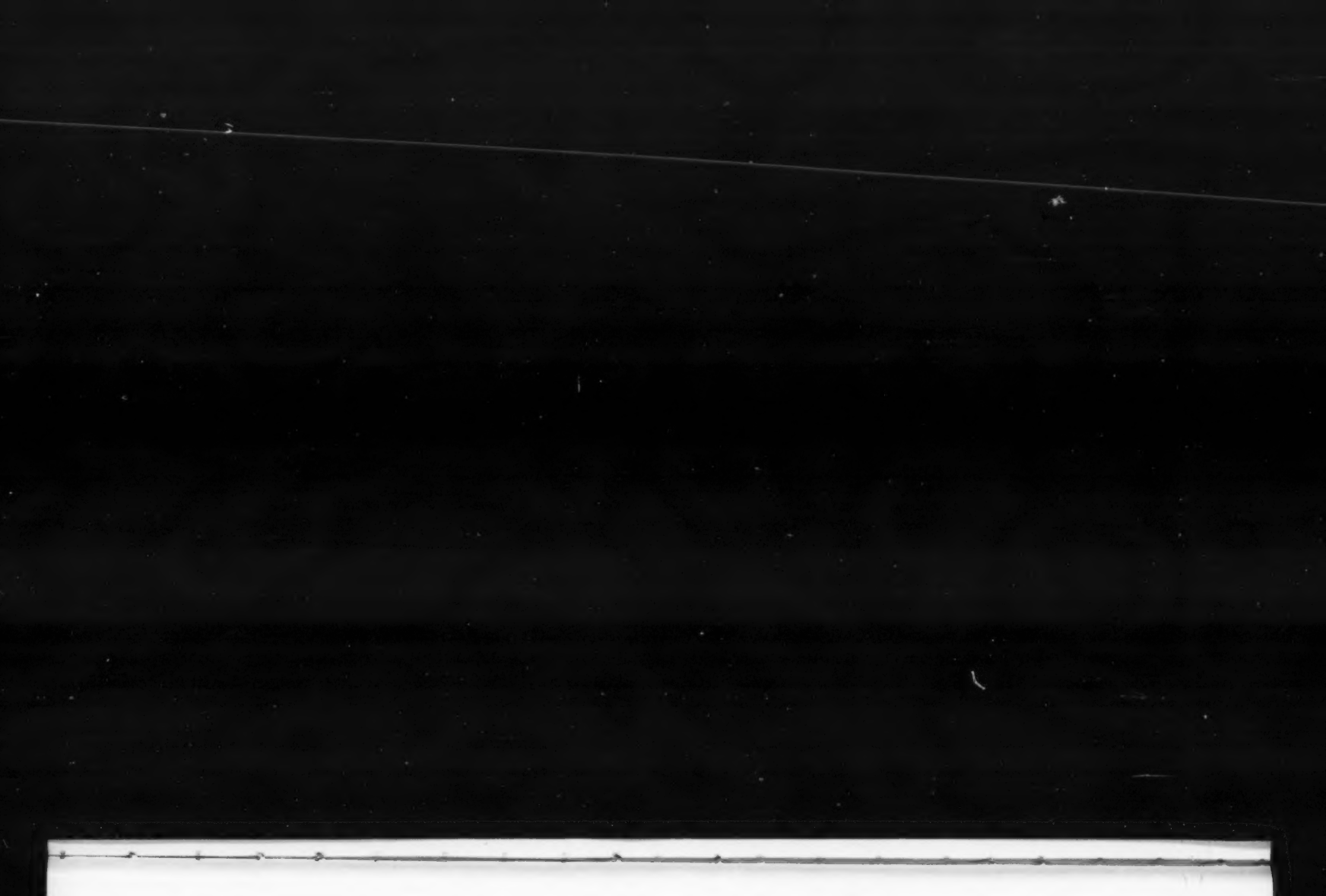

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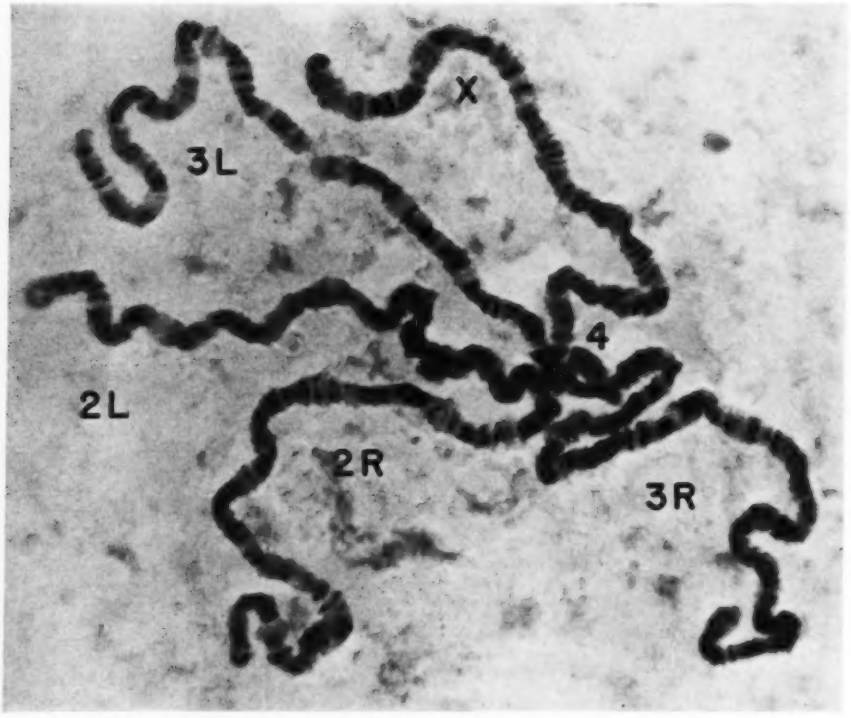

THE SALIVARY GLAND CHROMOSOMES OF DROSOPHILA

Frontispiece

Salivary gland chromosomes, female *Drosophila melanogaster*. The chromosomes are all associated in the chromocentral region and are free of contact loops and overlaps. All have the normal or wild-type sequence of bands except the *X*-chromosomes which carry the d1-49 inversion. These giant chromosomes facilitate the study of the effects of X-rays on the chromosome.

INDUCED CHROMOSOME REARRANGEMENTS IN DROSOPHILA MELANOGASTER

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CHROMOSOME breakage may be induced by irradiation of the living cell. If the spermatozoa of Drosophila are irradiated, some of the resulting chromosomal alterations may be detected by cytological analysis of the salivary gland nuclei of first generation progeny of the treated fathers. Such an analysis, conducted in this laboratory to determine the distribution of X-ray induced breaks along the chromosomes of *Drosophila melanogaster*[1,17] (also current investigations), disclosed various types of alterations, including several complex chromosomal rearrangements. They form the basis of the present report, in which selected material is used to illustrate some of the types observed.

Males were irradiated at 1,000, 2,000, 3,000, 4,000, or 5,000 *r* units and mated with virgin females. Salivary glands secured from the first generation larvae just ready to pupate, were prepared for study by Bauer's modification of the aceto-carmine technique. This consists essentially in spreading the glands in aceto-carmine on an albuminized slide by pressure applied to a temporary cover, transferring to 95 per cent alcohol for dehydration and removal of the cover, and remounting in Euparal.

When the chromosomes of Drosophila are broken under the influence of X-rays, new combinations of sections may originate. Some such combinations apparently prove lethal, since mortality is high in egg stages (current studies in this laboratory). Other recombinations are viable and provide material for the determination of the positions of the breaks along the chromosomes. This analysis is facilitated by the generally close association of homologous sections of the salivary gland chromosomes. Pairing of non-homologous regions such as was reported by McClintock at pachytene in Zea[22] was never observed in these preparations. Any cytologically detectable changes induced in the chromosomes contributed by the irradiated fathers can be recognized, therefore, by comparison with the normal wild-type maternal chromosomes present in the same diploid nucleus. By these methods the various rearrangements herein described have been detected.

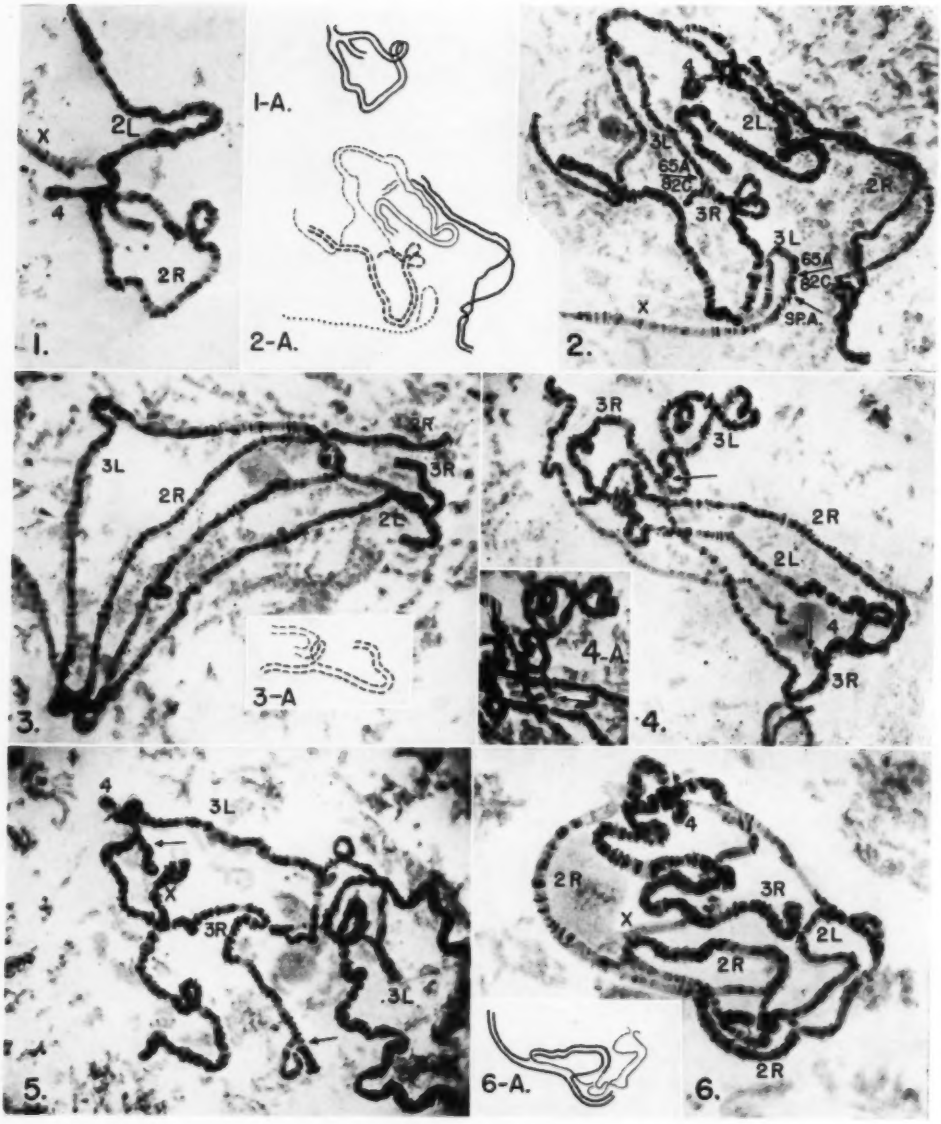
Types of Rearrangements

Observed chromosomal alterations include deficiencies, duplications, inversions, translocations, and various combinations of these involving two or more chromosomes. A photograph of salivary gland chromosomes which are all associated in the chromocentral region and which are free of contact loops and overlaps is presented in the Frontispiece as a basis for comparison with the deranged chromosomes shown in Figures 1-19.

The Intrachromosomal Types

Deficiencies—All observed deficiencies represent intercalary losses, as for example, the sequence *abdefg* derived by loss of *c* from the normal *abcdefg*. The feature permitting ready diagnosis of a deficiency in a salivary chromosome is the loop caused by bowing out of the normal, maternal homologue opposite the deficient section. This type of pairing is represented in Figures 5 and 7. In these cases, however, the section shown as deficient is not missing but is inserted elsewhere in the paternal set.

Very few small deficiencies were discovered in this material since to do so would require critical band-to-band searching along the lengths of all the



CHROMOSOME REARRANGEMENTS DUE TO X-RADIATION

Figures 1-6

These photographs show inversions and transpositions brought about in salivary gland chromosomes of Drosophila by X-ray treatment of sperm. This treatment causes breaks in the chromosomes. When the broken pieces are assembled in a different order, the chromosomes present an abnormal appearance when paired with the primary arrangement, due to the band-by-band somatic synapsis of homologous parts in salivary gland material.

chromosomes. Such deficiencies which occur in irradiated cells can be identified most easily if they are associated with known phenotypic effects by which their approximate location can be determined.[7,29,30] The longest deficiency observed extended from 10*B* to 11*D* in the X-chromosome (location of breakage points was determined in all cases from Bridges' 1935 maps[2]).

In certain complex alterations involving duplications, one of the two duplicated paternal chromosomes may be deficient for a long section. This type will be described subsequently.

Inversions—These likewise were all intercalary, and frequently intrabrachial: *abedcfg•hijklm* compared with the normal *abcdefg•hijklm* (the dot between *g* and *h* represents the spindle attachment region). The shortest inversion encountered in the present study included about 25 bands, which paired nevertheless with their homologues in a high percentage of the nuclei to form a small, although characteristic loop. Such a loop, although for a much longer inversion, is shown in Figure 1. It results from pairing between the normal right limb of the second chromosome (*2R*) and one with an inverted section extending from 41*B* in the chromocentral region to 59*B*. A series of similar figures describing inversions, especially in the *X*-chromosome, have been recorded by Hoover[12]. Inversions in the second and third chromosomes frequently encompass parts of both limbs as shown in Figure 2. When such inverted sections pair with the normal chromosome, they form a configuration which may be regarded either as a large inversion loop which extends across the spindle attachment region, or as an interchange crossover between two arms of one chromosome. If the two limbs of the chromosome become separated in the chromocentral region, as frequently occurs in pressing the glands for study, the loop configuration is destroyed.

Some glands revealed several inversions per cell, two or more in the same chromosome limb, either independent of each other (such as *acbdfeg•hijklm*) or in tandem (*acbedfg•hijklm*). The participation of inversions in complex rearrangements will be described later.

Transposition is the term here applied to an intrachromosomal translocation.

Symbols Used in the Illustrations

The symbols *X*, *Y*, *2L*, *2R*, *3L*, *3R*, 4 on photographs refer to the different chromosomes and their right and left limbs. In the drawings (*1A*, *2A*, *3A*, *6A*, *14A*, *15A*, *18A*, *19A*) *2L* is represented by a light solid line, *2R* by a heavy solid line, *3L* by a light dash line, *3R* by a heavy dash line, 4 by a wavy line and *X* by a dotted line. The spindle attachment region is shown by *sp. a.* In Figure 19*A* the nucleolus is shown in outline. Figures 5, 13, 15 are from females, all others from males.

The writer is indebted to Rachel Parker Craig and Ruby Gay Stewartson for preparation of these plates.

Legend to Figures 1-6 Continued

Figure 1. Inversion in *2R*. Inverted section extends from 59*B* to ca. 41*B* in chromocentral region. *1A* shows details of pairing.

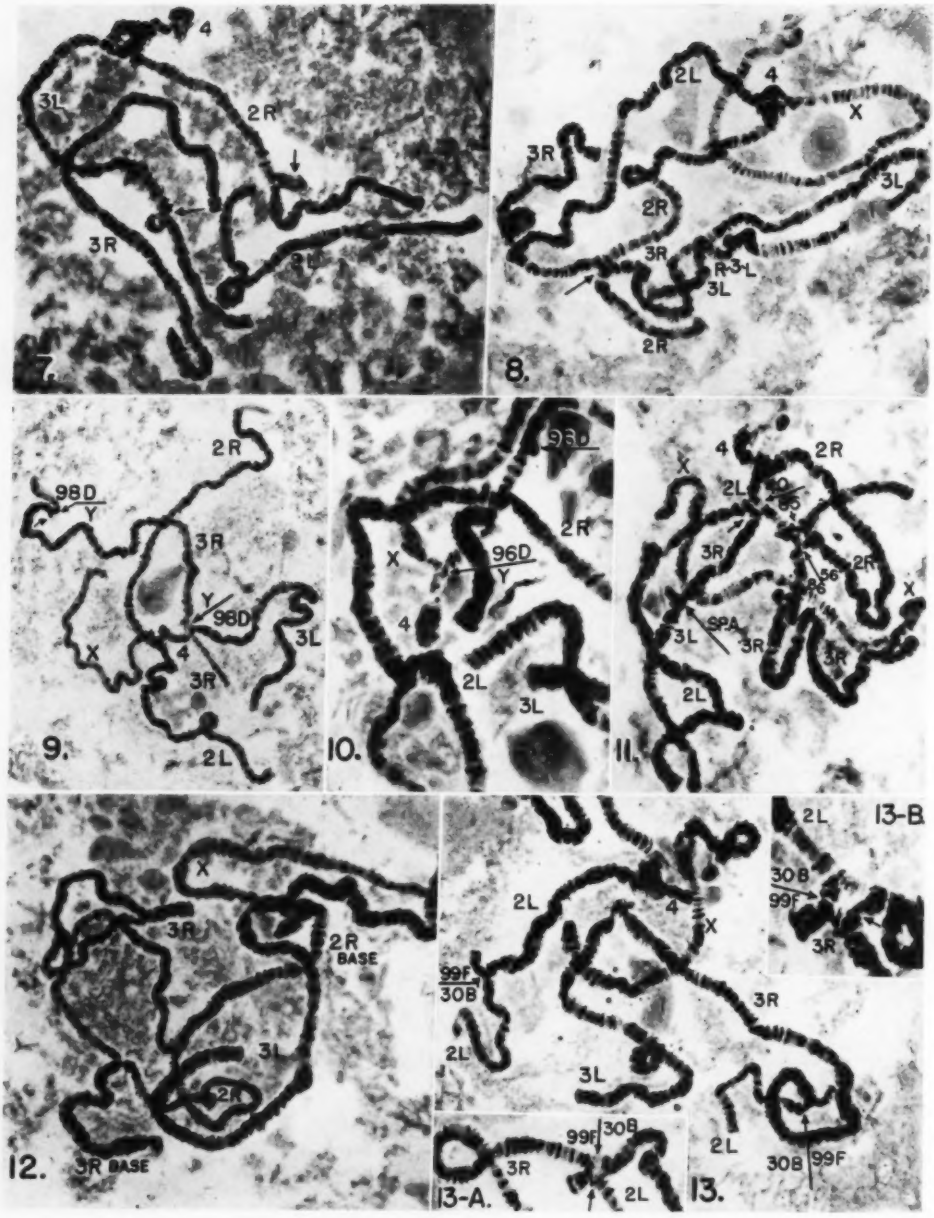
Figure 2. Inversion in third chromosome extending across spindle attachment region. Limits of inversion (65*A*——82*C*) indicated by arrows. *2A* shows details of pairing and identifies the different chromosomes.

Figure 3. Transposition of piece of *3R* (94*A*——95*D*) into 61*D* of *3L*. Pairing between the intercalated piece and the homologous region of the maternal chromosome detailed in *3A*.

Figure 4. Transposition of large portion of *3L* (61*C*——80*B*) into 90*B* of *3R*, as indicated by upper arrow. Lower arrow points to tip of *3L* (61*A*——61*C*) attached to 80*B* of chromocentral region of *3L*. Arrow in *4A* points to tip of unaltered, maternal *3L* not shown in focus in Figure 4. Reorganization of third chromosome: 100*F*——90*B*/80*B*——61*C*/90*B*——sp. a.——80*B*/61*C*——61*A*.

Figure 5. Transposition in *3R* and inversion in *3L*. Transposed section (96*C*——99*F*) inserted in chromocentral region (81) of *3R*. Lowermost arrow indicates deficient section; uppermost arrow indicates loop formed by piece inserted in chromocenter. Inverted section of *3L* not paired in this nucleus. Third chromosome reorganized as follows: 61*A*——64*F*/68*A*——64*F*/68*A*——*sp. a.*——81/96*C*——99*F*/81——96*C*/99*F*——100*F*.

Figure 6. Transposed section of *2R* (46*D*——53*E*) inserted in 25*D* region of *2L*. *6A* shows details of pairing.



TRANSLOCATIONS DUE TO X-RAY TREATMENTS

Figures 7-13

Figure 7. Simple (non-reciprocal) intercalary translocation. A piece of 3*L* (65*F* ..... 67*E*) is inserted in 51*B* of 2*R*. Arrow to left indicates deficient strand; arrow to right indicates

Transpositions were found in all chromosomes except the fourth. Pairing of transposed sections with homologous regions of maternal chromosomes is shown in Figures 3, 4, and 6; in Figure 5 these regions remain unpaired. The transposed piece may be quite long, as is shown in Figure 4, where a section extending from 61*C*-80*B* and including nearly all of the left limb of the third chromosome (3*L*) is intercalated in the 90*B* region of the right limb of the third chromosome (3*R*). The transposition of heterochromatin of the *X* chromosome to euchromatic regions where nucleoli then develop, has facilitated identification in the salivary glands of the nucleolus-forming region of this chromosome.[15]

Interchromosomal Rearrangements

Translocations—All simple translocations were of the intercalary type, resembling therefore the transpositions. Figure 7 shows such a non-reciprocal translocation in which a section of the third chromosome from 65*F* to 67*E* is inserted in the 51*B* region of the second chromosome.

The reciprocal translocations or interchanges between two chromosomes of the set constitute one of the most frequent types of induced alterations. Figures 8-13 show considerable variability in the degree of pairing between the altered and normal chromosomes in different nuclei.

Interchanges in which one of the participating chromosomes has been broken in the heterochromatic region require special consideration. Analysis of such cases is complicated by chromocentral association masking the limits of the individual chromosomes, and by the tendency of chromosome limbs to separate from each other at the spindle attachment region under the pressure of smearing. Thus in two reconstructed chromosomes *abcdefg*/wvu•tsrqpon and zyx/• *hijklm* (resulting from breaks between *w* and *x* in a chromosome *nopqrst•uvw-xyz*, and between *g* and the spindle attachment in the heterochromatic region of chromosome *abcdefg•hijklm*), compact chromocentral association of the regions adjacent to *g* may mask the *g*/w sequence, and not infrequently the chromosome may sever at this level. Likewise, if the two limbs of the zyx/•*hijklm* chromosome become separated, the place of attachment of the zyx/ • limb is no longer clearly marked. In cases of this type diagnosis is facilitated by analysis of haploid strands of the altered chromosome when present.

There is another type of translocation between euchromatic and heterochromatic regions in which a terminal section of

Legend to Figures 7-13 Continued

loop formed by section intercalated in 2*R*. Second and third chromosomes reorganized as follows: 21*A*—*sp. a.*—51*B*/67*E*—65*F*/51*B*—60*F*; 61*A*—65*F*/67*E*—*sp. a.*—100*F*.

Figure 8. Reciprocal translocation 2*R*-3*R*. As indicated at tip of arrow, pairing at interchange points (55*F* and 88*E*) is so complete as to simulate an overlap.

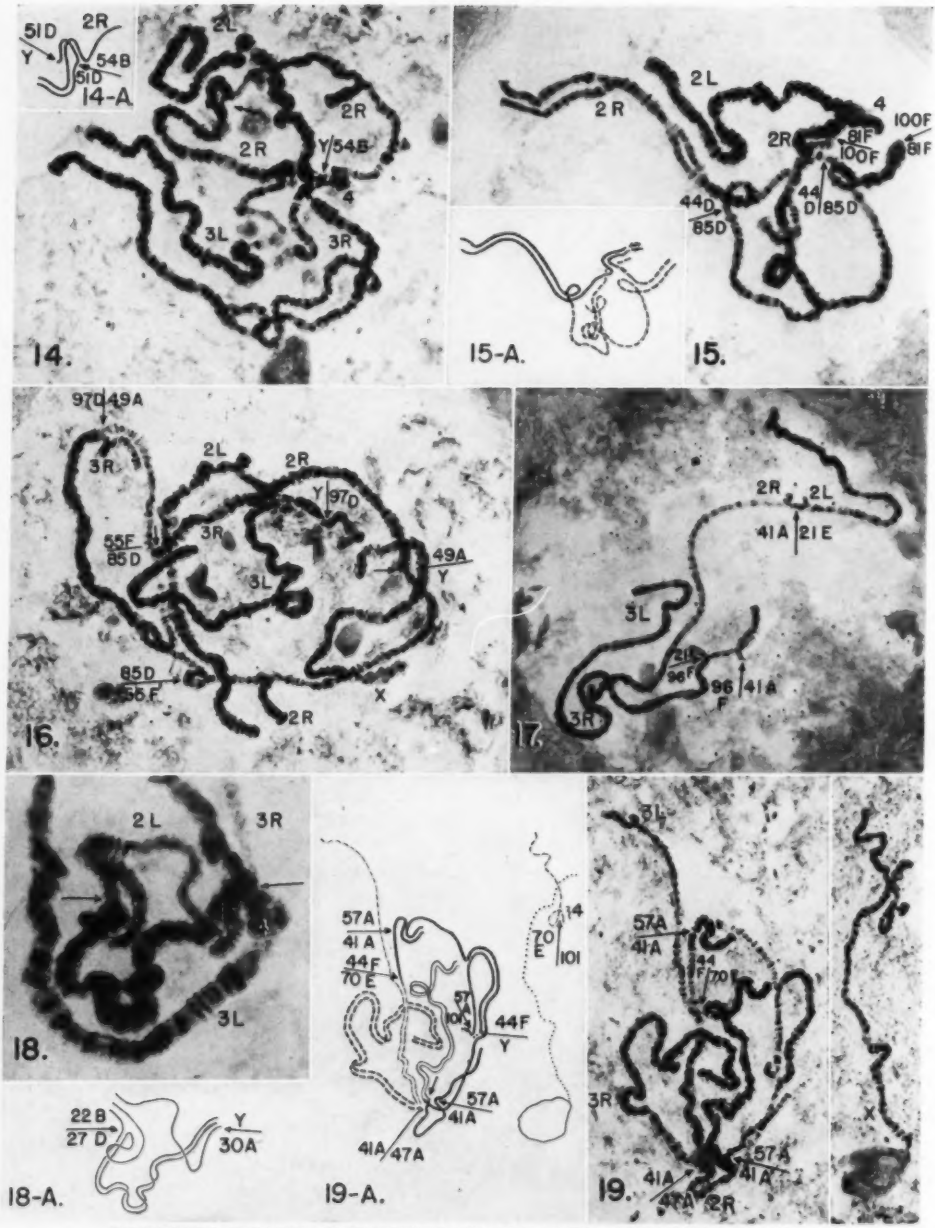
Figure 9. A 3*R*-*Y* reciprocal translocation. Break in 3*R* in 98*D* shown by uppermost arrow. Note small nucleolus formed by distal section of translocated arm of *Y*-chromosome at this position. Distal section of 3*R* (100*F*—98*D*) is attached to proximal portion of *Y*-chromosome which is located in chromocentral region.

Figure 10. A 3*R*-*Y* reciprocal translocation. Break in 3*R* in 96*D*. Pairing shown between proximal regions of *X*- and *Y*-chromosomes (lower arrow). Upper arrow indicates distal portion of translocated arm of *Y* attached to 3*R* in 96*D*.

Figure 11. Reciprocal translocations: 3*R*-2*L*; 3*R*-2*R*. Exchanges between 40*C* and 85*C* are represented in the upper cross-shaped configuration, and between 56*F* and 86*A* in lower cross-shaped configuration. Second and third chromosomes reorganized as follows: 21*A*—40*C*/85*C*—*sp. a.*—61*A*; 60*F*—56*F*/86*A*—85*C*/40*C*—*sp. a.*—56*F*/86*A*—100*F*.

Figure 12. Reciprocal translocation 2*R*-3*R*. Breakage points in 56*B* and 87*E*. Translocated chromosomes paired in proximal and distal but not in intermediate regions. Note that tip of 3*L* overlaps translocation configuration.

Figure 13. Reciprocal translocation 2*L*-3*R*. Strands not paired distal to translocation points. Compare with 13*A* and 13*B* showing close pairing.



COMPLEX REARRANGEMENTS IN X-RAYED SALIVARY GLAND CHROMOSOMES

Figures 14-19

Figure 14. Multiple exchange involving inversion in 2*R* and interchange with *Y*-chromosome. Inverted section extends from 51*D* to 54*B*. Detail to left of arrow in photograph shown

a chromosome is persistently attached chromocentrally even when the translocated chromosomes remain unpaired, suggesting that the proximal part of one of the longer autosomes is not involved. In males this type of rearrangement has frequently been diagnosed as involving the *Y*-chromosome (Figure 9). Pairing of *Y*-chromatin with the proximal part of the *X*-chromosome, as in Figure 10, indicates that the *Y*-chromosome participates in the translocation. However, *X*-*Y* pairing does not invariably occur, or is not conspicuous, as Figure 9 testifies, so that the terminal section may appear to be attached elsewhere than to the proximal part of the *Y*. Another clue in diagnosis is sometimes offered by the location of a nucleolus adjacent to the breakage point in the euchromatin, suggesting transfer of the nucleolus-forming region of the *Y*-chromosome to that place (Figures 9, 16).

In the females, translocations similar in appearance probably involve those proximal heterochromatic chromomeres of the *X*, or even the fourth chromosome, which represent short limbs of these chromosomes. Precise identification of such rearrangements can not always be made from salivary chromosomes, but mitotic chromosomes afford material by which any appreciable translocation to the *X* or fourth chromosomes can be measured. Therefore, in recent experiments neuroblast cells have also been used to check questionable salivary gland diagnosis.

Two or more independent translocations may exist within a nucleus. Such translocations, both involving 3*R* (3*R*-2*L* and 3*R*-2*R*) are shown in Figure 11.

Multiple Exchanges—Other rearrangements involve mutual exchanges among sections adjoining three or more breakage points. For example, if the breakage points and adjoining sections are represented by *A*/*A*′; *B*/*B*′; *C*/*C*′, the reunion may be *A* with *B*′, *B* with *C*′, *C* with *A*′. Figure 16 shows such a rearrangement of parts of the second, third and *Y* chromosomes. When two of three breaks occur in one chromosome, the separated parts may recombine to give an inverted section, one end of which is translocated with another chromosome. This is illustrated in Figure 14, in which the 51*D* end of an inversion, extending from 51*D* to 54*B*, is translocated with the *Y*-chromosome. Similarly, Fig-

Legend to Figures 14-19 Continued

in 14*A*. Second and *Y*-chromosomes reorganized as follows: Proximal part of *Y*/54*B* ..... 60*F*; 21*A* ..... *sp. a.* ..... 51*D*/54*B* ..... 51*D*/distal part of translocated arm of *Y*.

Figure 15. Rearrangement involving 2*L* and 3*R*. Translocation break in 85*D* of 3*R* lies within an "inverted section" extending from 81*F* ..... 100*F*. 3*L* and *X* not shown in this figure. Details of pairing are shown in 15*A*. Second and third chromosomes reorganized as follows: 21*A* ..... *sp. a.* ..... 44*D*/85*D* ..... 81*F*/100*F* (tip); 61*A* ..... *sp. a.* ..... 81*F*/100*F* ..... 85*D*/44*D* ..... 60*F*.

Figure 16. Multiple exchange involving 2*R*, 3*R*, *Y*. Five breakage points are represented, at 49*A* and 55*F* in 2*R*, at 85*D* and 97*D* in 3*R*, and in the *Y*-chromosome. 55*D* and 85*D* participate in a reciprocal interchange; 49*A*, 97*D* and *Y* in another exchange. Nucleolus to right is organized by the nucleolus-forming region of the *Y*-chromosome. The second, third and *Y*-chromosomes are rearranged as follows: 21*A* ..... *sp. a.* ..... 49*A*/distal part of translocated limb of *Y*; 61*A* ..... *sp. a.* ..... 85*D*/55*F* ..... 49*A*/97*D* ..... 85*D*/55*F* ..... 60*F*; proximal part of *Y*/97*D* ..... 100*F*.

Figure 17. Multiple exchange. Inversion in 2*L*-2*R* and translocation with 3*R*. Second and third chromosomes reorganized as follows: 60*F* ..... 41*A*/21*E* ..... *sp. a.* ..... 41*A*/96*F* ..... 100*F*; 61*A* ..... *sp. a.* ..... 96*F*/21*E* ..... 21*A*. The first of these gives a *J*-shaped chromosome, of which the longer limb includes the combined length of nearly all of the normal second chromosome, the shorter limb a part of 41*A*, and 3*R* from 96*F* to 100*F* (the tip). Because of the separation of the chromosome limbs at the chromocentral regions this relationship is not apparent in the photograph.

Figure 18. Duplicated sections of 2*L*. Photograph shows association of three strands at tip of 2*L*. One of the paternal chromosomes is deficient for the section between 22*B* and 27*D*; the other extends from the tip of 2*L* to 30*A* where it is attached to the proximal part of the *Y*-chromosome. Fuller description is in text. Detail in 18*A*.

Figure 19. Duplicated sections of 2*R*. Duplications extend from tip of 2*R* to 57*A* and from 41*A* to 47*A*. Additional description in text. Details of pairing in 19*A*.

ure 17 shows a translocation between 96*F* in 3*R* and one end of an inverted section which extends across the spindle attachment region of the second chromosome (from 21*E* to 41*A*). The translocation point in 85*D* shown in Figure 15 is within the limits of an inverted section extending from 81*F* to 100*F*.

More complex rearrangements have been found which involve mutual interchanges at as many as eight breakage points. In occasional preparations 12 or 13 breaks have been detected, but in such alterations it has been found extremely difficult to determine accurately the recombination of sections. Since it has not proved practicable to photograph the more complex alterations, a selected rearrangement is presented below,* using the symbol / to represent breakage and reattachment points, and *sp. a.* to represent the spindle attachment region.

Recognizable breaks occurred at ten places in four different chromosomes; one break in the *X* (in 15C), one in the fourth chromosome (in 101), one in 2*L* (in 26*A*), four in 2*R* (in 44*E*, 48*A*, 51*A*, 55*B*), and three in 3*L* (in 67*B*, 75*A*, 80*C*). Two of these ten breakage points (26*A* and 44*E*) bound an inverted section; the remaining eight are involved in mutual exchanges (48*A*/75*A*/101/15*C*/51*A*/67*B*/55*B*/80*C*/48*A*).

Duplications—A few of the glands studied (about one per cent) contained duplicated sections of various chromosomes of the complex. Figure 18 shows a duplication for the distal end of 2*L*. Of the three chromosomes which are conjugated at their tips, one is normal and maternal in origin, the other two represent altered paternal strands. Since one of these is deficient for the section between 22*B* and 27*D* and the other extends only from the tip of 2*L* to 30*E*, where it appears to have been translocated with the *Y*, the sections which have been contributed in duplicate by the irradiated sperm extend from the tip of 2*L* to 22*B*, and from 27*D* to 30*E*. The second, third and *Y* chromosomes of this arrangement appear to be organized as follows:†

The few bands at the tip of 2*R* distal to the 60*F* break could not be identified in the various nuclei studied.

A more complex rearrangement involving duplicated sections of the second chromosome is illustrated in Figure 19. The chromosomes are rearranged as follows:‡

No evidence of the presence of the dis-

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*21*A* ... 26*A*/44*E* ... *sp.a.* ... 26*A*/44*E* ... 48*A*/75*A* ... 80*C*/55*B* ... 51*A*/15*C* ... 1*A*
2*L* / 2*R* 2*L* / 2*R* / 3*L* / 2*R* / *X*

sp.a. ... 15*C*/101, 102*F*
X / 4

sp.a. ... 101/75*A* ... 67*B*/55*B* ... 60*F*
4 3*L* 2*R*

61*A* ... 67*B*/51*A* ... 48*A*/80*C* ... *sp.a.* ... 100*F*
3*L* 2*R* 3*L* 3*R*

---

†21*A* ... 22*B*/27*D* *sp. a.* ... 60*F*/81*F* ... 100*F*
2*L* 2*L* 2*R* 3*R*

21*A* ... 30*E*/ ... *sp. a. of Y* ...
2*L*

Distal part of translocated arm of *Y*/81*F* *sp. a.* ... 61*A*
3*R* 3*L*

---

‡60*F* 57*A*/41*A* 44*F*/70*E* ... *sp. a.* ... 100*F*
2*R* 2*R* 3*L* 3*R*

21*A* ... *sp. a.* ... 41*A*/47*A* ... 41*A*/57*A* ... 60*F*
2*L* 2*R* 2*R* 2*R*

102*F* 101/57*A* 44*F*/ ... *sp. a. of Y* ...
4 2*R*

61*A* ... 70*E*/101 *sp. a.*
3*L* 4

tal portion of the translocated limb of the *Y*-chromosome was discovered. Participation of the proximal portion of the *Y* has been inferred from pairing relationships. As represented in Figure 19, the 102, 101/57*A*.....44*F*/*Y* chromosome forms a loop; in other nuclei the 44*F* end is drawn into the chromocenter as is characteristic of *Y*-chromatin material. In this rearrangement the duplicated sections extend from the tip of *2R* to 57*A*, and from 41*A* to 47*A*.

In another publication[16] there has been described an intercalary duplication which involves an end-to-end arrangement of sister chromatids in the pattern of a reversed repeat.

Discussion

The types of alterations herein described illustrate the wide variety of cytologically detectable changes induced by X-radiation of the sperm of *D. melanogaster*. Prior to Painter's discovery[26] of the salivary gland method of diagnosis many of the less complex types of changes such as deficiencies, inversions and translocations had been recognized by genetic analysis, in some cases combined with cytological observation of mitotic chromosomes. On the other hand, the more complex rearrangements are less amenable to genetic analysis and therefore have largely escaped detection by such methods. Oliver [24,25] noted that in complex translocations of Drosophila several simultaneous breaks and reattachments could be involved, but the study of Dubinin and Khvostova[9] first indicated the multiplicity of such breaks and the possible complexity of the resulting rearrangements.

In the present material the number of determinable breaks per changed sperm ranged between two and twelve. It should be emphasized that the number observed may not in every case represent the maximum produced in the irradiated sperm. It is probable that breaks occur which cannot be detected cytologically, breaks which "heal" without visible loss of material to reestablish the normal sequence of parts. Catcheside[5] reports that observations on the ring *X*-chromosome of *D. melanogaster* indicate that if single breaks occur, the breakage ends do not fail to rejoin. There are also inter- and intrachromosomal changes restricted to heterochromatin which are inconspicuous cytologically because the bands of these regions are poorly defined and normally lie in contact with similar bands of other chromosomes.

Determination of the exact number of breaks responsible for certain of the rearrangements can be reached only when an understanding of the sequence of events leading to their formation is attained. By way of illustration, a transposition can be regarded as requiring three concurrent breaks. The study of Dobzhansky and Sturtevant[8] on inversions in natural populations of *D. pseudoobscura* suggests, however, that the same type of rearrangement could be attained by two independent, consecutive inversions. Thus, *abcdefghij*• is altered to *ab*gfedc*hij*•, and by subsequent reinversion of edc gives *ab*gfcde*hij*•, which would appear cytologically as a transfer of *gf* to a new position between *b* and *c*. To complete these two inversions four breaks would be required, two of which must occur in the same locus or adjacent loci (close to *c* in the illustration). Cytological analysis of several transpositions gave no evidence of independent breaks occurring in adjacent bands, as, for example, *abcdefghij*• being altered to *ab*gfedc*hij*•, and subsequently either to the overlapping inversion type (*ab*gfhcde*ij*•) or the included inversion type (*ab*gfdec*hij*•). The probability of consecutive, coincident, independent breaks may seem negligible, except that Grüneberg[11] and Emmens[10] have reported such a case in the reinversion of an inverted section producing the phenotypic effect "roughest". However, if a transposition represents a double inversion the bands of the transposed section should be in inverted order. They were not so arranged in four out of nine transpositions in which band sequence was analyzed. Therefore, double inversion is evidently not prerequisite to the production of this type of alteration. More-

over, among six cases in which more than a single inversion occurred in a chromosome, no overlapping inversions were found. Since they exist in nature[8] and require independent, consecutive breaks for their realization, the absence of this type among induced changes also points to the conclusion that such alterations occur by a single sequence of break and reattachment and not a series of such steps.

When several breaks per cell occur complex rearrangements become possible. The frequency of multiple breaks and attendant complex rearrangements appears from available data to be greater at higher than at lower X-ray dosages.[1] Viability of these complex rearrangements depends on the absence of unbalancing deficiencies and duplications, and the presence of mechanically fit chromosomes. Chromatids with two spindle attachment regions or fragments without spindle attachments will not survive the mitotic divisions occurring during ontogeny. Kirssanow[20] has suggested that a mechanism akin to crossing-over promotes the formation of such mechanically fit chromosomes and reduces the chances of non-viable combinations. Direct observations of dicentric and acentric chromatids in irradiated mitotic cells of various organisms indicates, however, that such combinations do occur,[3] and Sonnenblick[31] reports in a preliminary note that chromosome fragments have been observed in cleavage mitoses following irradiation of the sperm of *D. melanogsater*.

In none of the rearrangements studied had the tip or distal end of any chromosome assumed an intercalary position, nor had any of the regions of breakage become free ends. That the latter may occur by irradiation is shown by the terminal deficiencies of the *X*-chromosome reported by Demerec and Hoover.[7] In the recombination of sections, broken end unites with broken end. Simple terminal translocations of the type so frequently inferred from earlier genetic data (chromosomes *abcde*• and *fghij*• altered to f*gabcde*• and *hij*•) were not observed in this material. There were no terminal inversions such as occur in natural populations of *D. ananassae*[13,18,19] and which presumably might result from a single break with inversion of the section distal thereto. It is possible that the production of these types by irradiation requires a truly terminal alteration, one which causes no breakage and no band deficiency but sufficient chemical change to confer the capacity for reattachment to the tip. This end might then join with a broken end leaving an intercalary region to function as a new distal end, as occurs in cases of terminal deficiency. On this assumption induced "terminal inversions" and "terminal translocations" would depend on the effects of two independent changes rather than upon a single one.

A survey of numerous induced chromosome rearrangements such as are here represented affords information concerning organization of the salivary chromosomes.[14] Chromosomes which are broken close to the spindle locus reveal the extent of the heterochromatin belonging to the various limbs and confirm thereby the interpretation that the chromocenter is not an amorphous mass but a close approximation of heterochromatic portions of the component chromosomes. Transfer of heterochromatin of the sex chromosomes to euchromatic regions where nucleoli then develop has facilitated identification in the salivary glands of the nucleolus-forming regions of the *X*- and *Y*-chromosomes.

The duplications constitute another group of changes of cytological importance because of their significance with respect to problems of chromosome structure. If the duplication which has been described as involving breaks in 41*A*, 44*F*, 47*A* and 57*A* in the right limb of the second chromosome is considered, it will be seen that there are several ways in which the alteration might have arisen. Assuming that breakage at each locus is the resultant of an independent change, the following may be considered as possible alternatives: (1) At the time of irradiation the second chromosome consisted of two closely apposed chromatids as the result of completion of its longi-

tudinal division. Both of the strands were severed concurrently at 41*A* and 57*A*, but separately and independently in the intermediate loci, 44*F* and 47*A*. (2) At the time of irradiation the chromosome had divided in part only and consisted of two strands in regions 44*F* and 47*A*, but of a single (undivided) strand in the proximal section 41*A*, and in the distal section 57*A*. Upon completion of the longitudinal division, one strand had breaks in 41*A*, 44*F* and 57*A*, the other in 41*A*, 47*A* and 57*A*. (3) At the time of irradiation the chromosome was represented by a single strand. Breaks occurred at the four loci indicated, and the resulting fragments were duplicated following the subsequent division of the chromosome. At the time of reattachment, "healing" restored the normal sequence in one chromatid in 44*F* and in the other chromatid in 47*A*. Breakage points served therefore as translocation points at 44*F* in one strand, 47*A* in the other, and at 41*A* and 57*A* in both strands.

The observed rearrangement does not aid in discriminating among these alternatives nor in determining whether any indirect effect of irradiation may be involved. It permits, however, in the case of alternative (2), a more precise definition of chromosome structure at the time of irradiation. For it is evident that if the assumption is made that a chromosome break implies an unsplit chromosome at the time of irradiation, and a chromatid break implies a split chromosome, then the right limb of the second chromosome had not split proximally or distally but only in the intervening distance. The nature of the experimental procedure here employed decrees that only cells existing as mature sperms at the time of X-ray treatment are used in fertilization. Occurrence of split chromosomes in the sperm of Drosophila was inferred by Patterson[27] from a study of the effects of X-radiation on the production of mosaic flies by breaks in the *X*-chromosome. These results suggest that some of the sperms (about one-seventh) carry split chromosomes. The present case permits, however, more critical definition of the time of splitting of the chromosome if the assumption is made that a chromosome break represents an unsplit chromosome at the time of irradiation, for it indicates that longitudinal division is either occurring or has been arrested during these stages when the sperm head is compact and visibly homogeneous. Moreover, splitting of the chromosome is not a process which is initiated distally and proceeds proximally zipper-like, or vice versa, but one which can occur in different regions at different times (cf. Darlington,[6] p. 426).

Available data, however, do not eliminate the possibility that if a chromosome is double at the time of breakage, both chromatids may be affected similarly, as suggested by Moore,[23] White,[32] Carlson,[3] Kaufmann.[14] In this event it is necessary to distinguish between structural and functional doubleness.[21] Published data of X-ray experiments are certainly not unequivocal with respect to this matter, and pending decisive experimental data the question of a distinction between structural and functional states with respect to response to irradiation must remain open.

Since writing the preceding paragraphs there has appeared an article by Sax[28] in which such experimental evidence is presented. Sax's finding that both chromosome and chromatid breaks occur at metaphase and anaphase from 7 to 72 hours after raying Tradescantia microspores, indicates that a single hit can break both chromatids at the same locus.

Summary

Types of chromosomal aberrations detected by analysis of salivary glands of F_1 larval progeny of irradiated males included inversions, deficiencies, duplications, translocations, and various combinations of these involving two or more chromosomes. Selected rearrangements are described and their significance appraised with respect to current cytogenetic problems.

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CONGENITAL MALFORMATION OF HANDS AND FEET IN MAN

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CRIPPLING deformities are among the most tragic misfortunes which affect the human race. They are often due to accident or disease — the virus of infantile paralysis, the spirochete of syphilis, and the less definitely explained rheumatic and arthritic infections and inflammations count their victims by the millions. Another group of deformities is hereditary in nature. Many instances of this type of inherited misfortune have been published in this JOURNAL: polydactylism, bone fragility, symphalangism, and so forth. A long list of such inherited defects of the skeleton and appendages might be enumerated. Many of these, on the basis of published data, are inherited as simple dominant characters, the normal being recessive.

On a recent trip in the Bombay Presidency, the author encountered a striking deformity of the extremities which is apparently new, and which is definitely inherited. The defect was so striking as to attract immediate attention. Having read in past issues of this JOURNAL other accounts of inherited deformities of the hands and feet, the author suspected that heredity might be involved in this malformation, and decided to investigate the family of the deformed woman. From a study of four generations of the family living in an isolated Hindu community, it has been possible to construct a pedigree which appears to establish quite definitely the manner of inheritance of the defect. The study was facilitated by the fact that the family all lived in a single community, in which marriage was restricted to members of a particular sect. Former articles have indicated that inherited malformations of the hands and feet have on a number of occasions arisen by mutation, thereafter being inherited as a simple Mendelian dominant. The defect here reported is another instance of this same train of events.

Description

In the present case, both the hands and feet below the wrists and ankle joints are so completely deformed as to lose all resemblance to the normal extremities (Figures 20-22). On the hands, all fingers except the little finger are suppressed. Even it is not quite normal, and is improperly placed. It is slightly spindle-shaped at the tip, and below this is more or less uniformly thick instead of tapering like a normal finger. Although there are two joints on this finger, it cannot be stretched out straight, but generally remains slightly flexed at the tip (Figure 21), due to a defective distal phalangial joint. The nail formation is also abnormal.

There is some variation in the development of the palms. In some individuals, the palms are broader with an additional rudimentary ring finger. The metacarpal bones are present in such palms. In others, the palms are turned into a thick, pad-like limb with only the little finger present (Figure 21). The friction-skin patterns on these deformed palms are quite unlike the lines on the normal palms. In all cases, the wrists and the arm above it are quite normal.

The malformation of the feet is even greater. Just below the ankle joint, there is a rudimentary growth of an extra bony structure with a slight scar at the tip (Figure 21). A slight variation in the degree of development of this bony structure was noted in the right and left feet of a given individual, and there were considerable individual differences. Hence the variation in the development of this bony structure seems to be an

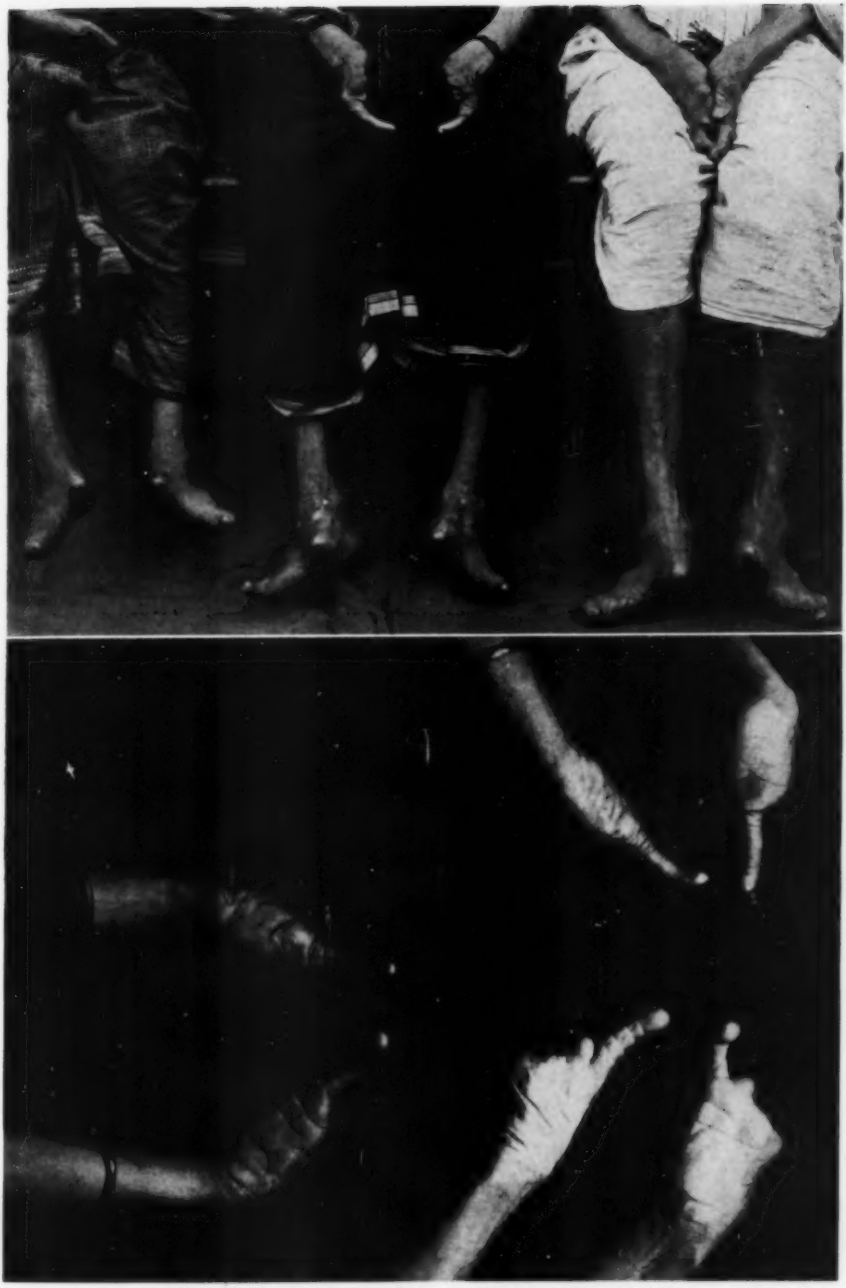
THREE GENERATIONS OF MALFORMED HANDS AND FEET

Figure 20

Daughters, mothers, and the grandmother on the right. The grandmother is V-14 in the pedigree chart (Figure 23) and the deformed daughters are VI-22 and 25. The abnormal child is VII-17 and the normal child is VII-14. The marriage of an affected individual who is heterozygous for the dominant gene would be expected to yield one normal child to one abnormal. As deformed parents thus have some normal children, they believed that the deformed children are born to them because of a lack of religious zeal.

individual attribute. The rest of the foot is deformed more or less into a flat-based conically-shaped organ with only one rudimentary toe at the tip, the remaining toes being completely suppressed (Figure 21). There is a slight development of a rudimentary nail on this single toe. The metatarsal bones of the foot are curved to a higher degree at the upper surface of the deformed foot than in the normal foot. The dermatoglyphic pattern on the skin of the plantar surface of the deformed foot is quite unlike the skin pattern on a normal foot. The ankle joint and the remainder of the leg are normal.

These people cannot walk normally. They invariably tilt to one side while lifting the leg, as they cannot balance well. They manage to eat with the aid



DETAILS OF THE MONO-DIGITAL MUTATION

Figure 21

Extremities of three women heterozygous for the dominant gene for malformation of hands and feet. Note the absence of normal toes and the extra bony structure below the ankle. On the hands the one finger present bears some resemblance to a normal little finger. On the broader palms a rudimentary ring finger may be seen. The friction-skin pattern on the malformed palms bear little resemblance to the normal. The arms and legs above the wrists and ankles are entirely normal, and the affected individuals are normal in all other mental and physical respects.

of the little finger and the deformed palms. Other personal requirements cause them much inconvenience. They are, however, quite normal in mental qualities and in other physical development.

Variation in Malformation

As already mentioned, there is some variation in the malformation of palms. Some members of these affected families have broader palms with an additional rudimentary ring finger (Figure 21), while the palms of the others are deformed into a thick pad-like structure with only the little finger projecting from it. Out of 23 cases studied, nine individuals had such broad palms with a rudimentary second finger. The palms of the remaining individuals manifest the extreme deformation, with a single finger. The review of these cases and a study of the line of inheritance (Figure 23) indicates that the degree of malformation of the palms of the children has no relation to the degree manifested by the affected parent. A similar situation was observed by Hefner[4] in his study of symphalangism: "the trait shows many variations, but such are not transmitted; that is, any degree of symphalangism may appear in the offspring regardless of the degree manifested by the parent." The bony structure of the foot, noted above, seems to behave similarly.

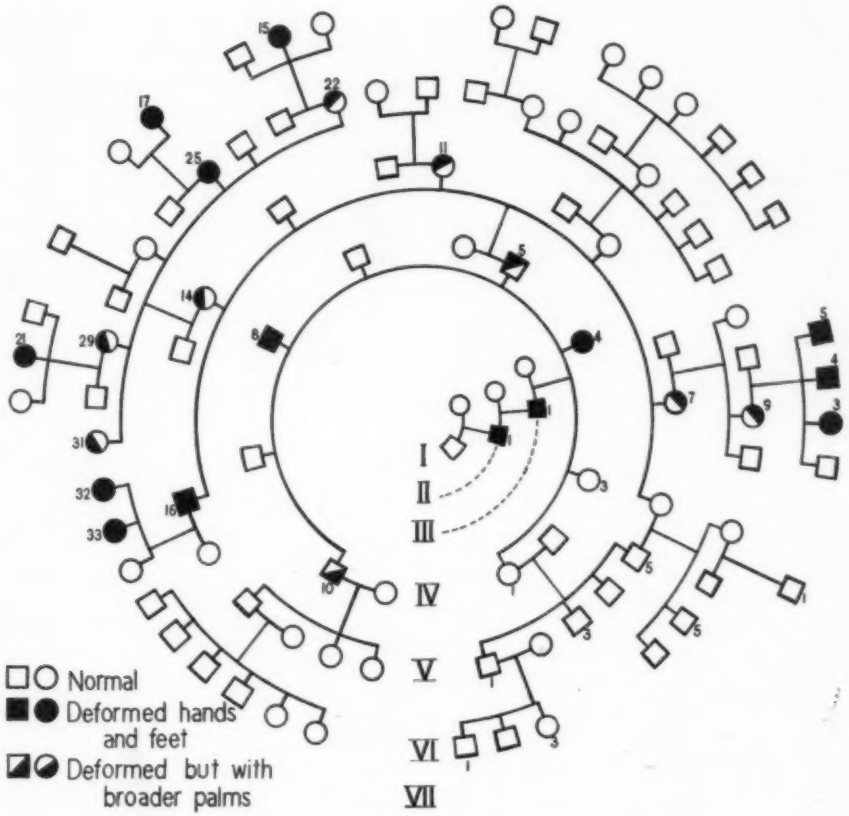
The affected woman whom the writer happened to encounter came from a Havic Brahmin community (Hindu) of the North Kanara District, Bombay Presidency, India. A genealogical investigation of her family through four generations revealed that this malformation of hands and feet is inherited as seen in the accompanying chart (Figure 23). The first individual who showed the trait was born to a normal couple. This boy lost his father soon after his birth and his mother did not marry again. In the second generation also there was only one child, a boy with malformed hands and feet. Out of eight children of this individual, four were deformed. Individual IV-10 had three children but none of them was affected.

MALFORMED MOTHER AND NORMAL CHILD

Figure 22

The same mother and child as in Figure 20, showing the grip of the mono-digital hand. Normal children such as this resulting from marriage of heterozygotes to normal (homozygous recessive) do not transmit the defect, as is seen in V-5 and 6 (Figure 23), both of whom came from affected families.

The members of these affected families believe that such children are born to them as a result of the curse of God, and that if certain laborious religious ceremonies are performed in propitiation, they will beget normal children. The



SIX GENERATIONS OF MALFORMED HANDS AND FEET

Figure 23

Pedigree chart showing the origin of the mutation from normal parents. Although in the second and third generations all offspring are affected, and in two families, IV-10 and V-11, all are normal, the chance errors are balanced if we total all offspring in affected families to date which give 23 affected to 23 normal, exactly what one would expect for a simple, autosomal Mendelian dominant.

normal family of IV-10 is attributed to his successful performance of this religious ceremony. What is more probable is that the few children born to him escaped the malformation of hands and feet through the chance distribution of chromosomes. As the father was heterozygous for the character, half of his children would be expected to carry the gene for the defect. Had he had a larger progeny, like his father, the defect would surely have been manifest in some of the children.

When a child is born with normal hands and feet, his children are all normal, even though the marriage takes place between normal people coming from related affected families. This is shown in the children of the cousins V-5 and V-6. This observation is evidence confirming our belief that normals are homozygous recessives.

This malformation came into existence through mutation, for it was not manifest among the kinsmen of the first affected individual. There is no earlier record of such a malformation in this Brahmin community, which is a closed population because marriage is strictly restricted to other members of this same sect. This fact helps substantiate the origin of the mutation. If the mutational view is correct, the first individual affected must have been impure or heterozygous for the malformation and as such he is expected to produce two kinds of offspring in equal numbers on marrying a normal woman. Unfortunately for our study, the father of the first individual died soon after the birth of his first child. In the second generation also there was only one boy, whom we assume to be heterozygous like his father. In the third generation the eight offspring of whom four are affected gives the expected 1:1 ratio. Further, when all the thirteen families in which one parent possesses this trait are studied collectively, it is seen that 23 have normal hands and feet and 23 are affected. This ratio is exactly 1:1, an observation which leads to the conclusion that this malformation is definitely inherited as a Mendelian dominant. Since all marriages of affected individuals are with normals, no homozygous affected persons have been observed. The deformity, disastrous as it is in the heterozygote, might be even more serious in a homozygous individual.

Modifying Factors

The variation in the degree of the malformation of palms and the presence or the suppression of the rudimentary ring finger seem to be due to modifications in the expression of the main factor, either through environmental agencies acting early in embryonic development or through the action of genetic modifying factors. Hefner[4] found similar variations in his study of symphalangism of the hands, which he explained as follows: "Such a factor must itself be capable of modification to produce the various aspects of the trait, since symphalangism presents a series of variations and even disturbance of the characteristic bilateral symmetry. It is thus a logical deduction to assume that the gene itself is an organism subject to changes which are here expressed as a variable trait. The nature of such changes and the reasons for their occurrence must be intimately associated with the primary cause of mutation itself." However, various degrees of deformity are to be explained, the existence of a simple Mendelian factor difference seems to be established.

Although in the first two generations only the male offspring manifest this trait and in the fifth generation only the females, both sexes are affected in the third, fourth, and sixth generations. Hence this trait seems to be autosomal rather than sex linked.

Conclusions

1. This trait of malformation of hands and feet came into existence by mutation.
2. It is a single factor mutation which affects only hands and feet. It behaves as a single Mendelian dominant in inheritance.
3. It seems that this trait is autosomal and not sex linked.
4. There is some variation in the degree of malformation; the offspring exhibiting any degree regardless of that manifest by the parent.

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THE INHERITANCE OF SILKY PLUMAGE IN THE DOMESTIC PIGEON*

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IN 1925 most of the literature on the silky or lacy plumage of certain varieties of pigeons was reviewed by Steele[5]. Two further valuable discussions are those of Hall[1] and Spruijt[4].

Steele made a study of breeding records obtained from Mr. F. E. Pollex, a breeder of white silky Fantails, in Toledo, Ohio. According to these records silkiness seemed to be sex-linked and recessive to normal. Steele himself made no breeding tests.

A pair of white silky Fantails was purchased from Mr. Pollex in 1927. In this laboratory the pair produced 23 young; 18 were silky and five normal. The normal offspring were typical white Fantails, and of both sexes. The silkies for the most part closely resembled the parents, but a few were markedly more silky than the parents; these two grades of silkiness we shall term moderate and extreme, respectively.

Since these findings were entirely at variance with the interpretation that silkiness is recessive and sex-linked, further tests were undertaken. Several varieties of normal pigeons were introduced, including Homer, Pigmy Pouter, Jacobin, and Archangel. All matings were confined to individual coops, and sex in all recorded cases was determined by breeding test or autopsy. In 15 matings of silkies (moderate grade) together, 104 offspring were obtained, of which 24 were normal and the remainder silkies of both moderate and extreme types. The ratio of normal to silky is very close to 1:3. Three pairs of such extracted normals in turn produced 21 squabs, none silky. Crosses of female moderate silkies with normal males gave 72 silky and 81 normal offspring, with the sexes about equally represented in each class. Similarly, male moderate silkies in crosses with normals produced 74 silky and 78 normal offspring. The results are summarized in Table I. All the evidence indicates that a simple dominant factor, not sex-linked, is responsible for silkiness.

The silkies of extreme type appeared only among the offspring of silky × silky matings. On this account it was assumed that they were probably the homozygous phenotype, but proof was not obtainable at first, as the Fantails of this type failed to produce any offspring. However, sterility is not a consistent feature of extreme silkies, since two, a male and a female, F_3 and F_2 respectively, from outcrosses, gave good fertility. The female, 2557 E, was mated to an Archangel; she laid ten eggs and raised nine young successfully, all silkies of moderate grade. This strongly indicates that she was homozygous. Further support for the supposition that the extreme silkies are homozygotes is also found in the fact that no moderate silky has proved homozygous.

The results of our tests make it difficult to account for the data on which Steele reported. Dr. Steele has kindly placed at our disposal all the original records used in his table (1925), and a few others given him subsequently by Mr. Pollex. The later records include a significant mating, of two silkies (pair 20), which produced five offspring: two silky and *three normal.* No records of sex are given in the later data, and in the material published by Steele the sex of only ten offspring was proved by breeding. In view of the discordance of the later records with the

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NORMAL FEATHER COMPARED WITH TWO DEGREES OF SILKY

Figure 24

Magnified views of normal (A), moderately silky (B), and extremely silky (C), demonstrating the homozygous normal, the heterozygote, and the homozygous dominant silky respectively. In each view the shaft is at the left, with the barbs branching from it. In the heterozygous form the barbules tend to bend, an indication of weakness. The homozygous silky feather has a worn appearance and the barbules are disarrayed. The barbs are also uneven, resulting in a less clear focus in the photograph. All of the photographs are on the same scale.

SILKINESS DUE TO BARBULE DEFECTS

Figure 25

Wing covert feather of normal (*ll*), moderate (*Ll*), and extreme (*LL*) silky. Silkiness reduces strength and elasticity of the barbules, and there is a tendency for the barbs to twist.

earlier, and the small numbers involved in those matings that have critical value, we can safely discard the conclusions based on them in regard to the mode of inheritance of silkiness.

It is probable that most silky pigeons trace their ancestry to birds of the same type known centuries ago. That the trait may arise occasionally as a separate and independent mutation is shown by a case that recently came under our observation at the Palmetto Pigeon Plant, Sumter, S.C. The breeding tests were made under the supervision of one of us (W.F.H.), and we are able to make use of the records by the courtesy of Mr. Wendell M. Levi, president of the plant.

A single silky male offspring appeared from a mating of a male white Carneau with a female blue Homer, both with normal plumage. Among many subsequent offspring no similar squabs were found, and silkiness had never been known in either parental breed. The sport was first mated with an entirely unrelated Homer. Six young were obtained, of which four were silky. He was next mated with an unrelated white Carneau and produced four squabs, one of which was silky. This proves that the mutant was heterozygous for the dominant factor, as would be expected. Crosses have not yet been made with other silky stock, but the fact that the character is in every way similar and is also a simple dominant makes it highly probable though not certain that the same gene is involved.

Structure of Silky Feathers

That silky feathers fail to web normally is readily apparent (Figure 25). A study of the structural basis of silkiness was first made by Steele[6]. He noted that, unlike the condition in the case of the silky fowl, the barbules do not lack hooks; general weakness, fragility, and frequent disarray of the barbules, and a tendency of the barbs to twist seemed to be the only definite abnormality. The appearance of feath-

MALE SILKY PIGEON

Figure 26

Photograph of black silky male 2596 *B*, showing the effect of the dominant gene for silky feathers in the heterozygous condition. This pigeon has pearl colored eyes.

ers under moderate magnification is in agreement with Steele's observations (compare Figure 24*B* and *C* with Figure 24*A*). Teasing the barbules with a needle shows not only that they are weak, but that their elasticity is very poor, especially in the extreme silky.

Steele believed at least part of the trouble to be due to a relatively excessive number of barbs in the silky feather. However, we have counted the barbs on 5-cm. lengths of tail feathers from ten normal and ten silky specimens, and cannot confirm Steele's conclusion. Both the highest and the lowest values were found in the normal feathers, and the averages were nearly identical, viz., normal 166.7, and silky 164.

Silky squabs may often be recognized before the feathers emerge, as the nestling down tends to curl. Here again the extreme silkies show the more obvious effect of the gene.

Silky plumage is by no means limited to white Fantails, or even to the Fantail breed. Spruijt[4] figures a number of silkies, most of which appear to be extreme and lacking the characteristic features of the Fantail. Onink[3] shows a white silky Fantail which also had feathered feet and a crest.

We have obtained combinations of silky with several of the known unit factors in pigeons, e. g., crest, pearl iris color, ash-red plumage color, chocolate-brown color, checker pattern, and solid black. All the combinations were readily recognizable. In Figure 26 is shown a white-spotted black silky male with pearl iris, 2596 *B*.

Black plumage color, as contrasted with the wild-type blue, was shown to be a simple dominant by Jones[2]. It was given the symbol *S* (spreading factor). The male in Figure 26 and a sister are heterozygous for *S* and silky, in the coupling phase. These two birds have been briefly tested for linkage. The results (Table II.) show that if silky and *S* are linked, they are certainly not close together.

Another linkage test was made for silky with pure white plumage color. Although the factorial basis of white is not yet clearly understood, it seemed that in this case a single factor differentiated the normal stocks used from the white Fantail, as all F_1 were moderately pied and simple segregation was observed in F_2 and backcrosses to the white Fantail. The results of this test

(Table II.) also show no evidence of definite linkage.

TABLE I. Tests of Inheritance of Silky Plumage of Both Grades.

Number of Matings	PARENTS ♂	PARENTS ♀	OFFSPRING SILKY ♂	OFFSPRING SILKY SEX ?	OFFSPRING SILKY ♀	OFFSPRING NORMAL ♂	OFFSPRING NORMAL SEX ?	OFFSPRING NORMAL ♀
15	SILKY (moderate)	SILKY (moderate)	38		42	9		15
Total				80			24	
19	SILKY (moderate)	NORMAL	41	2	31	30	2	46
Total				74			78	
23	NORMAL	SILKY (moderate)	34	8	30	43	10	39
Total				72			81	
Grand total		obtained: expected: (1:1)		146 152			159 152	
3	NORMAL (from silky)	NORMAL (from silky)		0			21	
1	SILKY (extreme)	SILKY (moderate)		3 *			0	
1	NORMAL	SILKY (extreme)		9			0	

* 1 extreme

TABLE II. Linkage Tests of Silky with Solid Black and with Pure White.

Number of Matings	PARENTS ♂	PARENTS ♀	OFFSPRING NON-CROSS-OVERS	OFFSPRING CROSS-OVERS
1	BLACK SILKY	BARRED NORMAL	5	6
1	BARRED NORMAL	BLACK SILKY	7	3
Total			12	9
4	PIED SILKY	WHITE NORMAL	5	12
7	WHITE NORMAL	PIED SILKY	33	26
Total			38	38

Conclusions

Silky plumage of the types here studied depends upon a single factor, dominant to normal, and not sex-linked. The moderate grade is the heterozygous form, and the extreme grade is apparently the homozygote. We suggest that the symbol *l* proposed for silkiness by Steele be altered to *L*.

The effect of the factor is apparently a reduction in strength and elasticity of the barbules, and a tendency of the barbs to twist. The down of nestlings also tends to curl.

Combinations with several known factors have been produced and easily recognized. Linkage of silkiness with the factor for solid black or with pure white probably does not exist

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PLANT HORMONES IN 1937

IN reviewing a monograph one can only refer to some details in order to illustrate the general impression which the book makes. Indicative of the usefulness of the monograph under review,* the reviewer has used the book as a reference for a course in Growth and Development together with that of Boysen Jensen (translated by Avery and Burkholder, 1936). These two publications were used partly because of the material they present, and partly because the many personal views expressed by Boysen Jensen are equilibrated by the personal views given by Went and Thimann.

The value of this monograph would be still greater if it had not been published shortly after Boysen Jensen covered the same ground. One of the authors seems to have realized this because he has written a general review

*Went, F. W., and K. V. Thimann. *Phytophormones*. Experimental Biology Monographs. XI + 294 pp., 61 Figs. Macmillan Co., New York. 1937.

again in 1938. However, thorough acquaintance with the content of the monograph by all the workers in the "phytohormone" field is highly advisable since some of the work recently published is virtually repetition of work mentioned in this book!

The authors state that they have added many new experiments for the sake of completeness, but on the other hand they do not give a detailed and critical treatment of 77 publications which they cite as appearing in 1936. Inasmuch as the reviewer has over 40 additional articles dated 1936, the views expressed in the book are not necessarily based on a complete survey of existing data.

The reader will find two chapters of great practical use, namely that on "The Technique of Auxin Determinations," which serves as a comprehensive compendium for any beginner in the field and contains a valuable reduction of the various units used to one standard, and the chapter on "Chemistry of the Auxins" which gives a clear description of the status of this field.

Since the monograph is indispensible as a source of a great many facts, and will therefore be widely used, the reviewer should like to illustrate the danger of overemphasis which is apparent throughout the whole book by one example, namely the transport of auxins. On page 14 is mentioned that "among many remarkable facts concerning the transport of auxins in the coleoptile, particularly the strict polarity of its movement has been demonstrated." On page 99 we learn that "it has of course been known for a great many years that any substance, even though it be toxic, when it is once in the vessels will be carried with the transpiration stream," and on page 100 that "the normal transport of auxin is through living tissues and it is this transport which is polar." As a matter of fact the transport in the coleoptile as a whole is not polar as is stated on page 12, but only that part which goes through the living tissue. However, on page 101 we read that "considering the auxin transport in shoots when the applied concentration exceeds a factor of 100 times or so, the polarity disappears," presumably because this concentration is no longer physiological. Moreover, on page 147 is stated that (cf. page 97) considering the root, "there seems no doubt that transport in both directions occurs in Pisum, etc." The statement on page 12 must therefore be limited (a) to living cells according to page 100, (b) to living shoot cells only according to page 147. Nevertheless the fact that the auxin transport is polar, is emphasized throughout the whole book and not the evident and necessary qualification that the transport is polar or non-polar dependent upon the tissue concerned.

On page 98 is stated that "in 1926 Went had concluded from the effect of light on auxin curvatures that light decreases the rate of auxin transport." Two sentences later is stated, after referring to some experiments of the reviewer which proved this contention, that "one can only say with certainty that light appears to have no effect on auxin transport." On page 171, however, we see that although "light has no effect on the longitudinal transport," in any case a change in lateral transport is proven to be caused by light, but "the way in which unilateral light brings about this lateral transport is not understood." Thus the statement of page 98 that light changes the transport at large which is contradicted on the same page, is again contradicted and affirmed on page 171. Even if one considers that the authors sometimes refer to lateral transport and at other times to longitudinal transport without keeping the distinction clear, one finds that their general conclusions show again the tendency to emphasize a certain viewpoint. They do this also in respect to the effect of light on longitudinal transport. Data not given, but referred to on page 98, are said to show that light not only causes a lateral transport, but also changes the longitudinal transport of auxin,—if one is careful in the choice of the experimental conditions. On ac-

count of some negative results reported by others, the authors state on page 98 that "the polarity of auxin transport is difficult to influence from without." On page 171 again they state that "light has no direct effect on the longitudinal transport" and that the explanation of the effect of light "based on decreased rate of transport on the lighted side, cannot be valid." Thus the ambiguous statement on page 98, that it is difficult to affect the longitudinal transport, which was dependent on the balancing of positive results against negative results, is changed in the explicit statement on page 171 in favor of the negative results, although based on the same references.

Similar examples could be given on the problem of root growth and especially from other chapters on the physiology of the auxins all of which would demonstrate that the interpretations given in the book are by no means settled, and that therefore the workers in this specific field still have to go back to the original literature.

The book makes excellent reading for the non-specialist reader, since he will perhaps be inclined to agree with the authors and will be delighted to learn that (page 248) "many problems have been solved and few really important points are still subject of disagreement."

The specialists have again to decide on the old question of preference between an objective discussion, which is bound to be somewhat dry, and a more subjective approach which seems inevitably doomed to be somewhat one-sided, even if one takes into account the unquestionable competence of the authors.

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THE CASE FOR INHERITANCE OF SCHIZOPHRENIA

THIS book is rather difficult to review, because so much information is packed into its 272 pages of reading matter that to abstract it is almost to reproduce the book. It is not an easy book to read because of the constant mathematical analysis which runs through the pages, yet it is a book which is very worth while because of the constant endeavor to check all ideas and conclusions by examination of the figures concerned. To the exponent of eugenics who feels that sterilization will settle all eugenic problems, the book will be a disappointment, for it clearly points out that sterilization of the schizophrenics will lessen the incidence of schizophrenia very little, owing to the fact that schizophrenia develops often when the patient has already had most of the offspring which he or she will have. To the ardent exponent of environment and psychic trauma as a cause of schizophrenia it will also be an equal disappointment, because the author emphasizes the fact that all schizophrenia is hereditary, not only those cases in which an obvious family history of the disease exists, but also those in which there is no such history. The conclusions must be given without much comment upon the method of proof as this would involve too lengthy a review.

Half of Insane are Schizophrenic

The problem of Schizophrenia is important because it is the most prevalent form of mental disease found in insane asylums. It is called dementia praecox on this side of the Atlantic. The same disease by any other name is just as bad, only the psychiatrists like the new name better. One-fifth of all the beds in all the hospitals in the United States, so I have seen it stated, are occupied by dementia praecox patients so it is a tremendous problem. Kallman states that according to Landis and Page, it is responsible for 58.2 per cent of the residents of mental

The Genetics of Schizophrenia. By Franz J. Kallmann. Published by J. J. Augustin, N. Y. 289 pages text. 76 Statistical Tables. $5.00.

hospitals. Of 68,334 inmates of New York State Mental Hospitals, 36,504 are dementia praecox patients. Until the recently discovered shock treatment was available, no cure was known, and no treatment had much effect. It is too early to know whether the shock treatment affects permanent cures. Even if they are permanent, the social consequences of increasing the proportion of active and potential dementia praecox in our population could hardly be recorded on the plus side of the ledger.

The group which formed the basis of the study was composed of all the blood kin of those schizophrenics which had been admitted to a hospital in Berlin, between the years of 1893 and 1902 and whose clinical records were still available in 1929. Only those cases were included in which the diagnosis of schizophrenia could be considered as undoubted and in which the disease began before the 40th year. There were 1,087 schizophrenics, with 3,279 parents, husbands and wives, 3,384 direct descendants, 3,920 brothers and sisters and half-brothers and sisters, and 2,194 nephews and nieces. They represented a typical cross section of the population of North Germany, at a time when there was little if any practice of birth control. By virtue of the method of selection, the author states that a one-sided selection of late-developing paranoid schizophrenia was avoided. The material was divided into four diagnostic groups: (1) hebephrenia, (2) catatonia, (3) paranoid schizophrenia, (4) simple schizophrenia in which there was no marked tendency to mental deterioration, and only brief cycles of psychotic activity.

The first two groups he calls the nuclear; the last two the peripheral group. He finds this classification justifiable because of the differences between them which extend to a number of factors. The marriage rate and fertility rate of the nuclear group is much less than that of the average population, the marriage rate being one half the normal, and the number of children per psychotic patient little more than one. The peripheral group has a fertility rate which is hardly one third less than normal, averaging 4.2 children per proband. The unfortunate thing about the latter group from the eugenic standpoint is that 87 per cent of these children are born before the schizophrenia develops. In his material he found that approximately one third of the children of schizophrenic patients were born *after* the patient had developed the disease. Inasmuch as not all children born of a schizophrenic patient develop the disease, it is not certain that all of this third would have been schizophrenic. Hence while their birth may have been prevented, it would not have prevented an equal number of future schizophrenics from being born.

The author estimates that only 1.7 to 3 4 per cent of the total number of schizophrenics would be eliminated by sterilization of schizophrenics after their disease is manifest. He at once adds that in "thus establishing the inadequacy of sterilization of psychotic patients after disease-onset, we must avoid the usual error of accepting this as an argument against the introduction of eugenics on the whole. The need and practicability of prophylactic measures in the field of endogenous psychoses must be considered an exclusively medical problem dealing only with biological facts. It should not be confused by philanthropic, religious and philosophic considerations or complicated by political speculations and prejudices. Any pretexts advanced against eugenic prophylaxis are palpably insincere in a population which already practices birth control extensively for economic and social reasons. Granted birth control, it is difficult to understand why its first object should not be the prevention of inferior children."

He feels that schizophrenia has been proven to be an *organic* disease based upon a *specific* predisposition which follows a *recessive* course of heredity. From this it follows that one factor for it is transmitted to *all* the children of schizophrenics. Because of this recessive mode of inheritance, it may often be that there is no immediate history of schizophrenia in the ancestors of the patient, and it is often difficult to recognize

the carriers of the latent trait of schizophrenia. Therefore, it becomes rather difficult to apply a rational eugenic program to this problem.

That such a program needs applying is evident from a study of the type of the relatives of the probands apart from their schizophrenic tendencies. More than two-thirds of the children of schizophrenic patients, one-third of their brothers and sisters, nearly half of their grandchildren, and about a quarter of the half brothers and sisters, great-grandchildren, nephews and nieces are *undesirable* types in a community. So although it may not be possible to wipe out schizophrenia by sterilization of the schizophrenic patient, eugenic principles applied to all the members of a family in which schizophrenia has appeared would eliminate a large number of future citizens who are only a liability to the community. Although because of its recessive mode of transmission, schizophrenia does not always appear in the children of an affected parent, Kallmann states that the children of schizophrenics have a probability of becoming schizophrenic that is nineteen times that of the average population; while the grandchildren, nephews and nieces are five times as apt to develop the disease as is the average person.

There are apparently external factors that call for the expression of the schizophrenic trait. This is evident from the fact that not all children of two schizophrenic parents develop the disease, as they would if it were recessive and were conditioned by inheritance only. About 30 per cent of the children who have inherited factors which would make them schizophrenic do not actually develop the disease. This figure agrees with Rosanoff's statement that in about 30 per cent of identical twins in which schizophrenia develops in one member of the pair, only one twin is affected; the other remains free of the disease, although having the factors necessary for its appearance. He, therefore, urges that the marriage of an identical twin who is normal, but whose twin has developed schizophrenia, be prevented as the so-called normal twin is just as liable to transmit the disease as is the affected twin. He also points out that men or women whose first mate was a schizophrenic and by whom they had schizophrenic children should not remarry while they are in the reproductive period, for they themselves are carriers of the trait, as is shown by the fact that their children develop it.

Thus although Kallmann does not give us much hope of eliminating the disease through sterilization of the patients themselves; he would urge that all persons who are carriers of the trait; namely children who have had a schizophrenic parent, parents who have shown that they carry the trait obvious or latent as shown by the appearance of the disease in themselves or in their offspring, all relatives of schizophrenics who by virtue of their known genetic constitution are certain of their carrying latent a factor for the disease; and probably brothers and sisters who have an even chance of carrying it latent, even if they have not the disease itself should refrain from propagation, since all of them are potential transmitters of the disease itself.

(It is just at this point that the abstractor feels that Dr. Kallmann could have been a little more enthusiastic about the eugenic program for the schizophrenic patient himself. It may be that sterilization will not prevent at the most more than 3.5 per cent of future schizophrenics from being born; but as Kallmann points out, 30 to 35 per cent of the children of schizophrenics are born *after* their parents developed the disease, and therefore sterilization would prevent that same number of carriers from being born. It is from carriers that the new ranks of schizophrenics are recruited to such a large extent. Even if not a single schizophrenic was prevented from being born by sterilization of the patient with the disease, the prevention of a large number of carriers would be effected.)

Associated with Tuberculosis

There is no genetic correlation between schizophrenia and psychopathy or feeblemindedness, epilepsy, alcoholism or criminality. Where these conditions are found in the relatives of schizophrenic patients in excess of their amount in the

general population they are due to external factors. The exact mode of transmission of schizophrenia, whether due to a single recessive Mendelian character, or to a more complicated scheme, is not determined by his investigations. It is, however, likely that the transmission does not depend on two or more genes; and it is certain that it is recessive in character. Its final expression in the individual depends upon the total constitution of the person, which decides the form the disease is to take, and which is itself dependent upon heredity. There is a very close relation between the predisposition to schizophrenia and the low resistance to tuberculosis. Kallmann says that there is "such a startling correspondence between expectation of schizophrenia and mortality from tuberculosis that the ratio of frequency between the siblings and the children of probands and again between the children and the grandchildren, is exactly the same for the expectancy of schizophrenia and the mortality from tuberculosis." He thinks there is a genuine gene-coupling between the two conditions, and confirms the assumption that both are inherited as recessive traits. Their similarity lies in the hereditary inadequacy of certain tissues and he feels that this weakness is in the reticulo-endothelial system. This finding points the way to future clinical treatment, and substantiates the rational basis for that already devised, namely non-specific stimulation of this system by insulin, sulfosin, etc. The lines for future attack in psychiatry will be the prevention of the disease by preventing its hereditary transmission, and the healing of those cases which develop despite the preventive efforts.

The reviewer knows of no work dealing with the inheritance of schizophrenia which is so soundly based on observation; where conclusions are arrived at with so little coloring of prejudice, and where the worker has let his conclusions be dictated by fact and not by what he sets out to prove. The book is recommended heartily to psychiatrists who on the whole need the emphasis which is here placed on heredity; and to eugenists who may need the corrective influence of figures to prove that sterilization cannot accomplish everything they desire, because of the late onset of the disease. Both groups will find solace for their hurt feelings; the first because environment is admitted to play an inhibiting role in about 30 per cent of cases in which the disease might be expected to develop; the second, because the need for a rational eugenic program is clearly stated.

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A SURVEY OF GENE MUTATIONS

STUBBE presents in this issue of the "Handbuch der Vererbungswissenschaft" an exhaustive summary of our knowledge concerning the general features of the mutation process. The literature is included to the middle of 1937. A second part of the publication, which is promised for the near future, will be a catalogue of all mutations known among animals and plants. The present general account of the evidence relating to mutations opens with an interesting historical survey. Subsequent chapters concern the qualitative and quantitative features of mutations, and the final chapter deals with the nature of the gene. There is a literature list of 52 pages. On the whole, Stubbe has given us a much needed and very useful compilation. We may doubt whether it was necessary to present a great many interpretations which are no longer valid and which, at best, are only of historical interest Again, one might wish that some of the evidence, e.g. the facts relating to "pleiotropism," had been presented with more of a physiological approach. These, however, are minor faults in view of the obvious merits which an elaborate literature digest of the field of mutations must have in the eyes of all geneticists.

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*STUBBE, H., *Genmutation. I. Allgemeiner Teil.* Handbuch der Vererbungswissenschaft. Lieferung 23. 429 pp. 90 figures and 130 tables. Berlin (Gebrüder Borntraeger), 1938.

FAMILIAL INCIDENCE OF STAMMERING

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THE literature of speech correction is replete with statements by learned authorities both here and abroad attesting to their opinion that stammering is inherited.[1-5,7-9] There is no record in the literature, however, of any definitive attempt to prove this assertion. There have been studies made, notably those by Dr. Bryng Bryngelson at the University of Minnesota,[4] coupling the inheritance of stammering with the inheritance of left-handedness. Like other similar studies no controls were used for comparison and there was what appeared to be an attempt to prove an assertion already made. These studies have served to point a definite indication of the existence of an inheritable factor, however, and this study was made to prove or disprove as the case might be, the existence of familial incidence in stammering.

The incidence of stammering was examined in the backgrounds of 250 stammerers and a like group of non-stammerers (see Table I). The two groups were paired for age, sex, and social environment, within definite limits. The method used in selecting the families to be studied was to take the stammerers as they appeared in the clinics or as they were suggested to the author and pair them with completely unselected non-stammerers. The law of statistical regularity according to Jerome's *Statistical Methods*[3] reads, "that a moderately large number of items chosen at random from among a large number are almost sure, on the average, to have the characteristics of the larger group." This is the basis for most computations of social statistics being known as random sampling of the population.

In collecting the tabulated information concerning the family histories of the selected individuals the author personally interviewed the parents and solicited known facts concerning the ancestry. Wherever there was doubt, or one of the parents was without information, or wherever stammering was present in the immediate family and could be imitated, the record was not used. No attempt was made at finding predetermined information. The only facts elicited concerned the number of stammerers, their sex, and their position in the family tree. The results as here compiled, then, represent what the author believes to be reliable statistics concerning the incidence of stammering. If there be error, the assumption is that it is on the positive side rather than the negative, for the average person would deny rather than affirm the presence of stammering in the background as a matter of family pride. Family charts were made for each family investigated showing the position, age, and sex of each stammerer and their immediate families (Figure 27).

Cases for this study were obtained from the speech clinics at:

1. University of Chicago.
2. Central Free Dispensary in Chicago.
3. Michael Reese' Mandel Clinic in Chicago.
4. University of Wisconsin in Madison, Wisc.
5. The Public School System in Whiting, Ind.
6. The author's private office in Hammond, Indiana.

The following three tables represent, numerically, the accumulated data. Each table is composed of two parts, (a) the stammerers' backgrounds and (b) the non-stammerers', or the controls', backgrounds. Comparative ratios are shown with each table, one ratio showing the number of males to females and the other ratio comparing the number of stammerers found in the backgrounds of the two groups. Table II shows the same facts for the female stammerers' backgrounds and for their paired control non-stam-

TABLE I. Background of Male Stammerers and Male Non-Stammerers.

A. *Stammerers*	*Cases Studied*							*Total*	
1. Family type showing number of stammerers in each background	6	5	4	3	2	1	0		Mean 1.40
2. Number of families investigated	1	3	7	21	39	63	46	180	P.E. .077
3. Total stammerers found (1 × 2)	6	15	28	63	78	63	0	253	Ratio Male to
4. Female stammerers	2	3	5	12	18	11	0	51	Female 4 : 1
5. Male stammerers	4	12	23	21	60	52	0	202	
B. *Non-Stammerers*	*Cases Studied*							*Total*	
1. Family type showing number of stammerers in each background	4	3	2	1	0				Mean .211
2. Number of families investigated	1	3	3	19	154			180	P.E. .029
3. Total stammerers found (1 × 2)	4	9	6	19	0			38	Ratio Male to
4. Female stammerers	1	2	0	3	0			6	Female 5.3 : 1
5. Male stammerers	3	7	6	16	0			32	

Mean Difference 1.16
P.E. Difference .082

Ratio Mean A : Mean B as 6.63 : 1

TABLE II. Background of Female Stammerers and Female Non-Stammerers.

A. *Stammerers*	*Cases Studied*					*Total*	
1. Family type showing number of stammerers in each background	4	3	2	1	0		Mean .801
2. Number of families investigated	2	3	7	26	32	70	P.E.M. .089
3. Total stammerers found (1 × 2)	8	9	14	26	0	57	Ratio column
4. Female stammerers	1	2	4	5	0	12	5 : 4 as 3.7 : 1
5. Male stammerers	7	7	10	21	0	45	
B. *Non-Stammerers*	*Cases Studied*					*Total*	
1. Family type showing number of stammerers in each background	4	3	2	1	0		Mean .271
2. Number of families investigated	0	1	4	8	57	70	P.E.M. .027
3. Total stammerers found (1 × 2)	0	3	8	8	0	19	Ratio column
4. Female stammerers	0	0	2	2	0	4	5 : 4 as 3.75 : 1
5. Male stammerers	0	3	6	6	0	15	

Mean Difference .430
P.E. Difference .093

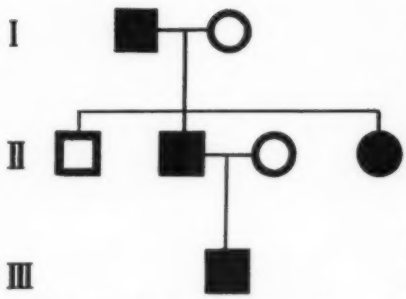
Chances in 100 of difference being a true difference — 100
Ratio Mean A : Mean B as 3.21 : 1

TABLE III. Composite Data for both tables.

A. *Stammerers*	*Cases Studied*							*Total*	
1. Family type showing number of stammerers in each background	6	5	4	3	2	1	0		Mean 1.24
2. Number of families investigated	1	3	9	24	46	89	78	250	P.E.M. 0.066
3. Total stammerers found (1 × 2)	6	15	36	72	92	89	0	310	Ratio
4. Female stammerers	2	3	6	14	22	16	0	63	5 : 4 as 3.92 : 1
5. Male stammerers	4	12	30	58	70	73	0	247	
B. *Non-Stammerers*	*Cases Studied*							*Total*	
1. Family type showing number of stammerers in each background	6	5	4	3	2	1	0		Mean .228
2. Number of families investigated	0	0	1	4	7	27	211	250	P.E.M. .012
3. Total stammerers found (1 × 2)	0	0	4	12	14	27	0	57	Ratio
4. Female stammerers	0	0	1	2	2	5	0	10	5 : 4 as 4.7 : 1
5. Male stammerers	0	0	3	10	12	22	0	47	

Mean Difference 1.012
P.E. Difference .067

Chances in 100 of difference being a true difference — 100
M A : M B as 5.44 : 1



A STAMMERER'S PEDIGREE
Figure 27

Pedigree of stammerer S. G., showing three stammerers in the background. Such pedigrees were constructed for each family investigated in both stammerer and control groups.

merers. Table III is a composite of the information shown in Tables I and II.

Evaluation of Data

Of the 250 families of stammerers studied, 172 showed some incidence of stammering, while 78 did not. A ratio of 2.2:1 or 68.8 per cent of the families showed the trait. In the non-stammerers families 39 showed some incidence of stammering whereas 211 did not, a ratio here of .18:1 or 15.6 per cent showing the disorder.

Statistically the results obtained are reliable, since a difference to be reliable must be at least four times its probable error. In this study the various differences were from four times to twenty times as great. Since it is also true that the reliability of a difference increases as the ratio of the difference to its probable error the possibility of pure chance creating the condition as noted is practically negligible.

The foregoing tables seem to express these pertinent facts: (1) that stammerers do tend to appear more frequently in the families of stammerers than in the families where there has never been stammering (in the ratio of 6.12:1) as has been predicted without statistical proof many times, (2) that stammering appears more frequently in the male than in the female (ratio of 4:1), a fact that is quite generally accepted. Dr. Robert West[11] in his summary of the White House Conference Report placed the ratios between the sexes at from 3:1 to 5:1; Dr. Travis[10] notes the ratios at from 2:1 to 10:1.

Conclusion

The data herein reported should lend weight to many considerations concerning stammering. For instance, that certain individuals in the general population are stammerers because of incidence of that disorder in their family backgrounds; that others are probably potential stammerers, but for some reason, at present unknown, do not stammer; that certain others, before this year is out, will become stammerers. It may be that stammering which is inherited duplex, that is, from both sides of the parental line, is permanent and cannot be successfully treated, or that stammering inherited, simplex, from one parental branch, is amenable to treatment. This line of thought can be extended almost indefinitely and further research is necessary before it would prove of value. It is true, however, that if stammering be inherited, treatment can effect only the present manifestation of the disorder and cannot effect the cause. In any case, the extent of our present knowledge is too meagre to allow more than conjecture at the present time. It is hoped that this study will provoke further research in stammering; that since the facts here gathered definitely indicate a true trend, such studies be integrated and the results of large numbers of family histories be published. The author does not wish to take a dogmatic stand as to the inheritance of stammering but considers the facts as gathered to be of importance and of wide interest. Any family histories of stammering will be received with deep appreciation.

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Who Discovered the Dominant Black Rabbit?

To the Editor:

In your issue for last January, p. 26, Mr. Aldous cites Dr. Schwarz to the effect that the mutation of steel-grey, or dominant black in rabbits, was first described and figured by Castle in 1925. It does not, of course, greatly matter genetically who discovered what and when, but in the interests of historical accuracy, I think I am right in ascribing the discovery of this mutation to Punnett. At all events, a description of it by Punnett appeared in the *Journal of Genetics*, in November, 1912, in a paper entitled "Inheritance of Coat-colour in Rabbits."

May I take this opportunity of saying how much I appreciate your admirable publication?

E. C. Richardson

Ecclesbourne, West Byfleet, England

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Biomathematics at Non-Genetic Levels

In some quarters there seems to be an impression that this book* will have a definite interest to geneticists, so it is not out of place to list its contents here. The book is divided into three parts. Parts II and III deal with excitation, conduction and similar problems of nerves. Part I and an appendix, together comprising one half of the total text, are concerned with a highly mathematical treatment of several aspects of diffusion, metabolism and mitosis rounded off with an eleven-page "Outline of a Physicomathematical Theory of Organic Form." Some of the results of this treatment may eventually prove to be of importance to the physiological geneticist, but probably not until they have been tested and developed by the general physiologist. This is apparently recognized by the author. Excepting a single sentence referring to the gene as a catalyst, genetics is not mentioned. The discussion of cell division is not primarily chromosomal and makes no reference to the type of problem investigated by Wrinch. These statements are not, in any way, meant to detract from the interesting ideas this book offers its intended readers.—R. C.

*Rashevsky, Nicolas. Mathematical Biophysics. Physicomathematical Foundations of Biology. Pp. xviii + 340. The University of Chicago Press, Chicago, Illinois. 1938.

A SECOND INDEPENDENT OCCURRENCE OF THE CURLY$_2$ MUTATION IN THE RAT

HELEN DEAN KING and LEON F. WHITNEY

HEREDITARY structural modifications of the hair, causing it to appear waved or curly, were not known to occur in the rat until a mutation of this type, *curly* (*Cu*), was discovered in 1928 in a male in the colony of wild gray Norway rats maintained at The Wistar Institute since 1919[3]. Subsequently two other mutations, phenotypically similar to curly but genetically different, were reported in this species: *kinky* (*k*) by Feldman[1], and *curly*$_2$ (*Cu*$_2$) by Gregory and Blum[2]. Suitable genetic tests have shown that curly and curly$_2$ are simple Mendelian dominants, and that kinky is a recessive. Mathematically the chances are extremely remote that one of these rare types of mutations would appear a second time in a genetically different strain of rats. This phenomenon has occurred, however, and is reported here, together with an account of the genetic tests made to establish its identity.

In 1930 the junior author obtained twelve albino rats from the Osborn-Mendel stock of Yale University. These rats formed the foundation for a separate colony that comprised about 300 individuals during a period of several years. As these rats were closely inbred, they became very uniform in various characteristics and were large, vigorous, prolific animals, apparently normal in all respects. In 1935 a litter was born in this strain in which one male developed a coat in which the hair was "woolly" in texture, and the vibrissae twisted or bent at the ends; the other members of the litter had normal coats and straight vibrissae. When this male was mated with a normal sister, half of the progeny had coats similar to that of the sire, and half were normal. This result indicated that the sire was heterozygous for a mutant character affecting the hair structure. Subsequent series of matings demonstrated that this was a dominant mutation, differing from normal coat by a single gene.

Several of these "woolly" rats were then sent to the Wistar Institute in order that the senior author might make the tests necessary to determine whether this mutation was a new type or a second occurrence of one of the two dominant curly mutations already known.

Homozygous "woolly" rats were mated with gray rats homozygous for *curly* (*Cu*). All of the 28 offspring obtained from these matings had curly coats which did not differ appreciably from those of their parents. These F_1 offspring were then used in two types of matings. Inbreeding F_1's produced seven litters containing 62 young, of which 30 were males and 32 females. Among these individuals there were 58 with *curly* coats and four that were normal. This finding accords closely with the distribution to be expected in the F_2 generation of a cross involving two autosomal factor pairs that segregate and act as duplicate genes. When F_1 males and females were mated with normal gray rats, the resulting 23 litters contained a total of 198 individuals of which 148 were curly and 50 normal, thus showing a ratio of three dominant to one recessive.

If these two mutations were identical, all of the F_1 offspring would have been homozygous for curly (*Cu*) and they should have produced only curly young in all types of matings made. The appearance of normal young in the F_2 generations furnishes conclusive evidence that the F_1 progeny were not homozygous and that the two mutations are genetically different, the genes producing them being located in separate chromosomes.

The *curly*$_2$ mutation was first discovered in 1933 in a colony of black hooded rats maintained by the Department of Anatomy of the University of California. Through the courtesy of Dr. Gregory, some of these mutants were sent to the senior author for genetic work, and this

stock was available for the further testing of the "woolly" mutation.

Mating homozygous *curly*$_2$ rats with "woolly" rats produced 19 young which were all curly, their coats having the same general appearance as those of the parental stocks. These F_1 individuals were either inbred or mated with normal grays. All of the 168 young obtained from inbreeding F_1's had curly coats similar to those of their parents, as did also the 92 young resulting from the second form of mating. As no normal young appeared in the F_2 generation of either of these crosses, it is evident that "woolly" is not a new mutation but a second occurrence of the mutation *curly*$_2$. The appearance of the same unusual type of mutation in two unrelated strains of rats is an event of such rare occurrence in mammals that we deem it worth recording.

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✦

A LINKAGE BETWEEN NAKED AND CARACUL IN THE HOUSE MOUSE

Charles B. Cooper
*Williams College**

In 1937 F. G. Carnochan[1] reported a new mutation in the house mouse, which was a peculiar coat character referred to as caracul. "The character is distinguishable from the time that the whiskers and body hair are visible. The body hair lies in fluffy waves which makes the mouse look much broader than the normal mouse, and the vibrissae are markedly curved. In the fourth week the body hair begins to straighten and the wave gradually disappears. . . . The coat when stroked the wrong way does not fly back as does the normal coat." Further investigation by L. C. Dunn[2] showed that caracul is dominant and viable in homozygous condition.

In the linkage experiments four multiple stocks were used, as follows:

N-stock *Nnt*^{1}*tWw* naked, fused tail, dominant spotting.

P-stock *aabbpps*$_e$*d*/*s*$_e$*d* non-agouti, brown, pink-eye, short-ear, dilute.

V-stock *ssd*$_2$*d*$_4$*w*$_1$*w*$_1$*v* piebald, leaden, wavy-1, waltzing.

W-stock *ffsh*$_3$*sh*$_4$*w*$_2$*w*$_2$ flexed, shaker-2, wavy-2.

These stocks, expressly bred for linkage work, greatly simplified the experiment, for fifteen chromosomes were marked with characters easily recognized in offspring. The *P*-stock was extracted following a cross involving a similar stock developed by Dr. W. H. Gates. The other three stocks were developed at the Roscoe B. Jackson Memorial Laboratory.

It was found that caracul (designated below by the symbol W_3) was closely linked with naked. The results were as follows:

Backcross Data (335 animals)

	W_3w_2nn	w_3w_2Nn	W_3w_2Nn	w_3w_2nn
F_1 ♂ × ♀ w_3w_2nn	140	119	4	2
F_1 ♀ × ♂ w_3w_2nn	29	41	0	0
Total	169	160	4	2

The crossover per cent was 1.8.

The caracul animals were kindly provided by Mr. Carnochan. The crosses were started and the problem suggested by Dr. George D. Snell, to whom the writer wishes to express his appreciation.

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*The work here described was carried out while the writer was a summer student at the Roscoe B. Jackson Memorial Laboratory.

POSTJUVENAL NUDE IN THE DEER-MOUSE*

FRANK H. CLARK
Edison Institute of Technology, Dearborn, Michigan

MATURE AND YOUNG NUDES

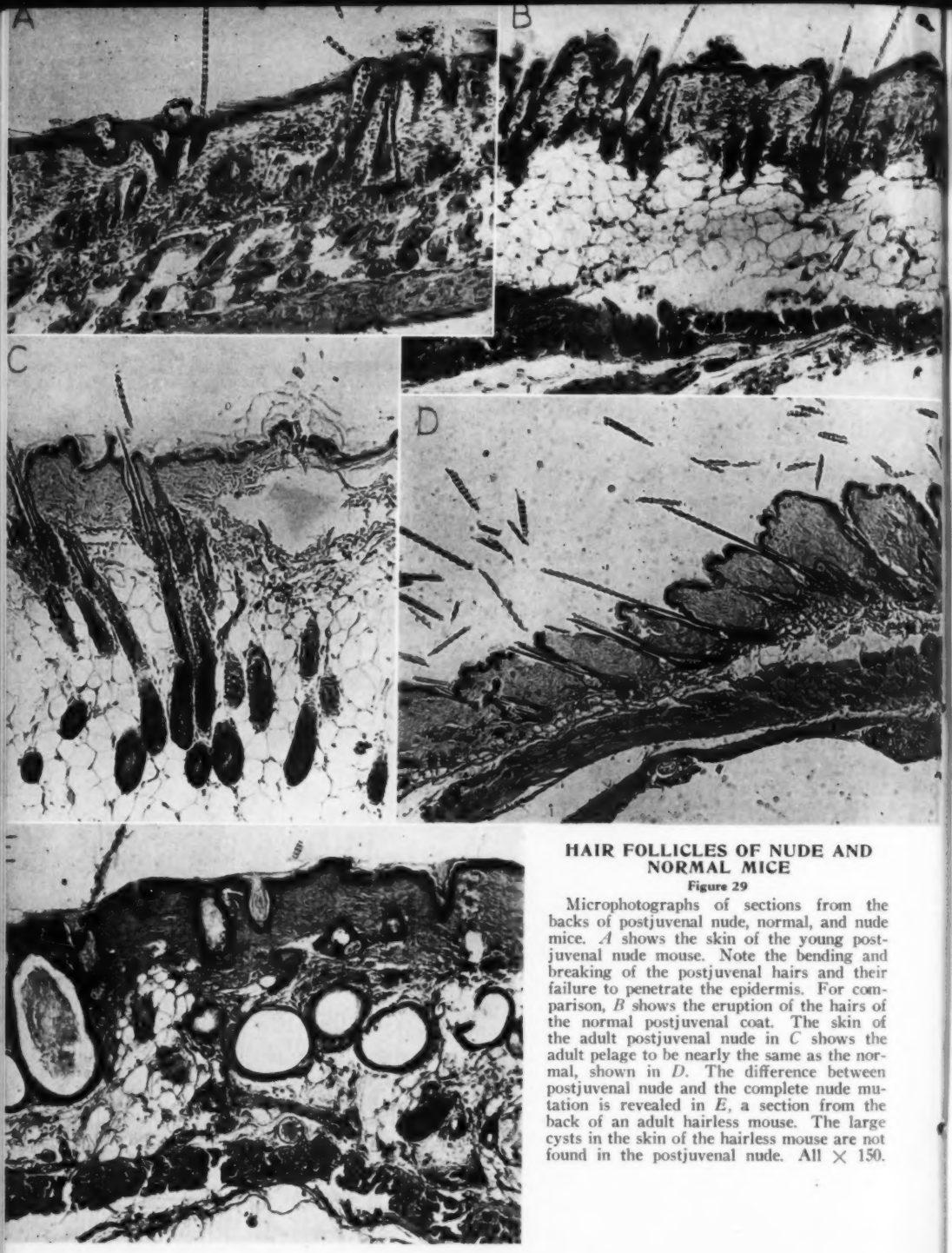
Figure 28

Photographs comparing an adult postjuvenal nude (*A*) and a postjuvenal nude mouse 29 days old (*B*). The nude condition is temporary and is caused by the breaking off and deterioration of the hairs of the postjuvenal coat beneath the epidermis. The mature mouse acquires a near-normal adult pelage, the nose and tail often remaining bare. The character is transmitted as a simple recessive.

TWO markedly different types of hereditary hair deficiency occur in the deer-mouse, *Peromyscus maniculatus*. The first type arose in the subspecies, *gambelii*,[2] and is similar phenotypically and histologically to recessive hairless mutations in the house-mouse and Norway rat. Degenerative changes in the hair follicles and sebaceous glands and lack of formation of the hair clubs[1] results in complete or nearly complete hairlessness at about 25 days

*Contribution from the Laboratory of Vertebrate Genetics, University of Michigan, Ann Arbor, Michigan.

The Photographs were taken by Dr. Lee R. Dice and the microphotographs were made by Donald Hayne.



HAIR FOLLICLES OF NUDE AND NORMAL MICE

Figure 29

Microphotographs of sections from the backs of postjuvenal nude, normal, and nude mice. *A* shows the skin of the young postjuvenal nude mouse. Note the bending and breaking of the postjuvenal hairs and their failure to penetrate the epidermis. For comparison, *B* shows the eruption of the hairs of the normal postjuvenal coat. The skin of the adult postjuvenal nude in *C* shows the adult pelage to be nearly the same as the normal, shown in *D*. The difference between postjuvenal nude and the complete nude mutation is revealed in *E*, a section from the back of an adult hairless mouse. The large cysts in the skin of the hairless mouse are not found in the postjuvenal nude. All × 150.

of age, a condition which persists throughout life.

A second hereditary hair deficiency, which I shall call *postjuvenal nude* recently appeared in the Laboratory of Vertebrate Genetics in the immediate progeny of a single pair of field-caught mice belonging to a stock of *P. m. osgoodi* Mearns collected by O. J. Murie near Bear Lake, Utah. This new mutation, as its name indicates, affects chiefly the postjuvenal pelage and has little or no effect on the juvenal or adult pelages. Deer-mice, homozygous for this defect, acquire a normal juvenal pelage but begin to shed this coat when they are between two and three weeks of age. Depilation of hair starts at the extremities and proceeds in a more or less diffuse manner over the entire body. The postjuvenal coat which normally grows in as the juvenal pelage is being shed fails to appear and the animals become completely nude at between 25 and 35 days of age, remaining that way for about two weeks. (Figure 28*B*.) During this stage, they are practically indistinguishable from deer-mice homozygous for ordinary hairlessness. When these mice are about 50 days of age, a new growth of hair begins uniformly over the body and this new adult coat when complete is normal in color and density. The tip of the nose usually, but not always, remains bare and the tail may or may not lack hair and the typical tail-stripe. An adult postjuvenal nude mouse whose nose and tail are both bare is shown in Figure 28*A*. Adult hairless mice may have more or less persistent or regenerated hair but it is always faded in color. This is probably due to the partial or complete degeneration of the hair follicles.

In crosses between nude and normal mice, the immediate offspring are completely normal. Matings between such F_1 animals have thus far produced 67 normal to 23 nude young, a deviation of only $0.5\pm$ a standard error of 4.11 from the 3:1 ratio expected in cases where a simple recessive Mendelian character is involved.

To make sure that postjuvenal nude is genetically distinct from ordinary hairlessness, nude and hairless mice were mated together. Twenty-five young have been born from these matings and all of them were normal. These results prove that nude and hairless depend upon different recessive genes for their expression.

Microscopic sections were made of the skin from the mid-dorsal region of nude mice when the hair was partly or completely shed. Sections were likewise made for comparison from normal sibs of these mice. A careful study of these sections indicates that postjuvenal nude is caused by a breaking off of the hairs of the postjuvenal coat at or beneath the surface of the skin (Figure 29*A*). This defect is apparently due to faulty keratinization of the hairs, and does not occur in the skin of normal sibs (Figure 29*B*). According to David[1] the partial hairlessness of the heterozygous dominant hairless (naked) house-mouse is due to a similar defect of part of the hairs. The hair follicles and sebaceous glands of adult nude mice appear to be normal (Figure 29*C*), and cysts similar to those found in the skin of hairless mice (Figure 29*E*) are never found. A section of the skin from the back of a normal mature deer-mouse is shown in Figure 29*D*.

Conclusions

Postjuvenal nude, an hereditary hair deficiency in the deer-mouse, *Peromyscus maniculatus* affects only the postjuvenal pelage and is a simple Mendelian recessive character. It is caused by faulty keratinization of the hairs of the postjuvenal coat, causing them to break off at or beneath the surface of the skin. Nude differs both genetically and histologically from ordinary recessive hairlessness in the deer-mouse.

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WATTLED PIGS IN PANAMA

DR. George O. Lee, of Canal Zone Junior College, Balboa Heights, C. Z., has recently addressed an inquiry regarding wattled pigs. He enclosed several photographs of the herd he has discovered in the vicinity of Panama. We are reproducing a few of these to show this variably distributed character, which is unknown in many regions.

The pigs are part of a herd owned by a Spanish speaking family of black Panamanian fishermen living at the mouth of the Santa Maria River in the Republic of Panama. The pigs show Poland China and Hampshire characteristics but are of nondescript breeding. The founder of the drove was a wattled female obtained from the mountains somewhere back of Rio Hato about ten years ago. Since then no outside stock has been added. Dr. Lee observed more than twenty head before the half-wild creatures scampered off through the bushes. About eight individuals in the herd did not have wattles.

The owners say that if neither parent pig has wattles, none of their offspring will have them. If either parent has wattles, most of the pigs of both sexes have wattles. These observations were not authenticated by Dr. Lee, but would indicate a simple dominant gene for wattles. One individual had a wattle on one side only.

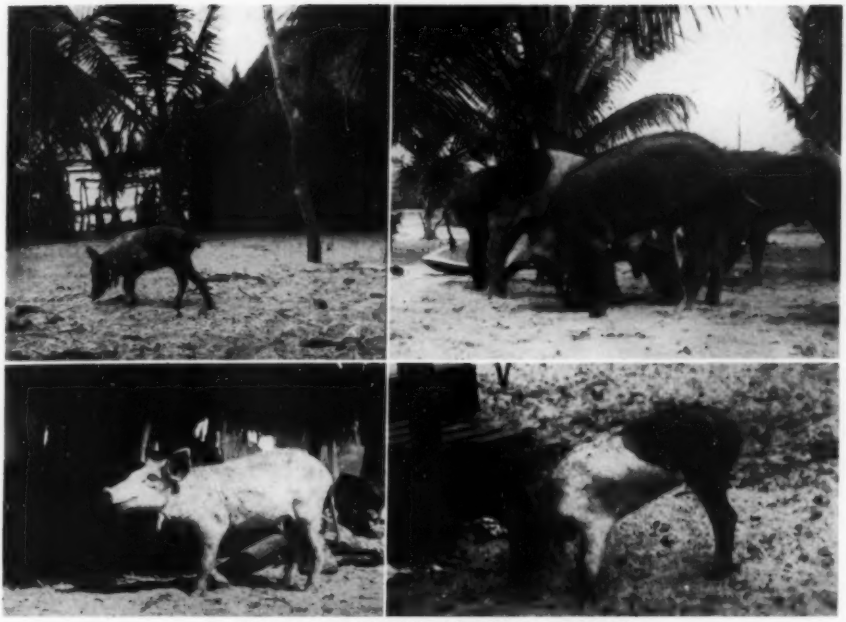
This character in pigs is one which is quite widely distributed in the southern part of the Atlantic States, and has also been recorded from Europe. J. L. Lush in this JOURNAL (17:73-91, 1926) discussed the inheritance of wattles in swine in connection with his observations regarding their inheritance in goats. The data there reviewed suggested that they were probably a simple dominant in inheritance, as were wattles in goats.

R. Froehner says that wattles "consist of skin and subcutis, and may also contain striated muscle, a piece of cartilage, blood vessels and nerves. In sheep they contain neither muscle nor cartilage." Bateson records one instance of a wattle containing bone. Their positions seem to be quite variable in different species, strains and individuals.

Morphologically the mammalian wattle seems to have little relationship to the wattle of poultry. The latter is considered to be a decorative secondary sexual character. It would be somewhat of a problem to rationalize a very compelling appeal into the more modest mammalian appendage. The size of wattles in poultry depends on the presence of the genital gland, as both wattles and comb of the capon are much reduced. No such relation has been reported in mammals.

The classic reference for wattles is William Bateson, who in *Materials for the Study of Variation* (1894) suggested a connection with the ear. He noted that "supernumerary auricles" had been observed on humans, swine, sheep and goats. At that time "neither cervical fistulae or auricles were known in the ox." The occurrence of wattle-like appendages in agnathic calves of the Kentucky Experiment Station herd as described and illustrated in the March, 1939, issue of this JOURNAL, appears to close the gap in the series. As these calves carried a lethal gene for jawlessness, the study of the inheritance of wattles in cattle appears to stop so far as this lead is concerned, as soon as it starts.

Bateson notes that in humans the presence of wattles "is generally associated with deformity of the external ear and often with aural fistulae." Such fistulae had been observed by Paget to occur in the ears of several members of a family, many of whom were affected by deafness. His information was adequate to show that the ailment was inherited, but did not suggest the manner. Bateson illustrated fully developed wattles on a child. One would hardly expect to observe these appendages on humans, even should they occur fairly often, as they



A WATTLE-BEARING HERD OF SWINE

Figure 30

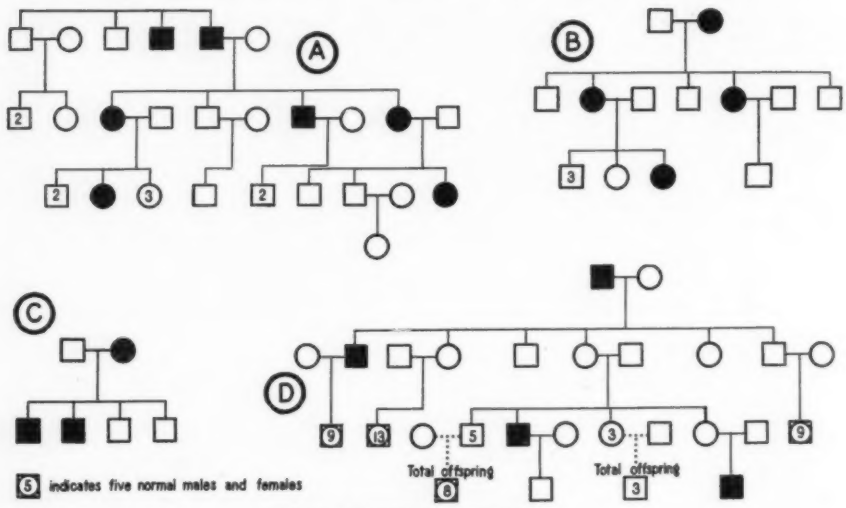
Photographs of some pigs found in Panama by Dr. Lee, which were part of a herd that had been closed to outside stock since the owners first bred the wattled female who was the founder of the herd. Ten years of free inbreeding have resulted in a herd in which a majority of the pigs are wattled.

can be removed by a simple operation, and would doubtless respond to tying off with a thread, much as small supernumerary fingers are often removed. Lush mentions the possibility of wattled sheep being phenotypically without wattles because of the likelihood of de-wattling in the shearing process.

The derivation and purpose of these appendages remain somewhat obscure. They seem to be analogous to the appendix, a hangover from what may once have been a useful organ. The source of the cartilage in wattles, according to Bateson, "may be parts of one or other of the usually undeveloped branchial arches behind the hyoid. As against this suggestion it is to be remembered that in the subsequent development of the neck those arches are pushed in far from the surface, whereas the cartilages in question are always superficial. Further, these repetitions have been known to occur at the openings of cervical fistulae, suggesting a comparison with the relation of the external ear to the hyo-mandibular cleft, but that such a relation to cervical fistulae is exceptional." His conclusion, and the assumption of more recent geneticists is "that these external projections, having the structure of the ear, must be regarded as partial repetitions of the ears." The absence of the hyoid bone in the wattled agnathic calves alluded to above may have some bearing on this speculation.

In recent studies of the inheritance of wattles, little work has been done on their morphology, and their possible purpose and derivation is still where it was when Bateson wrote his summary. They seem to have no purpose, nor has any linkage been found between them and any other trait.—R. C.-C. B.

INHERITANCE OF SHORT FINGER TENDONS



BENT FINGERS INHERITED

Figure 31

Pedigrees of four families carrying the gene for shortened finger tendons, which has usually been observed to behave as a dominant trait. In *D* the character seems to be recessive.

AMONG the many hereditary anomalies of the hands are tendons which are too short to permit normal flexion or extension of the joints.

PEDIGREE *A*

In this pedigree, that part of the extensor digitorum communis tendon inserted into the dorsal surface of the last phalanx of the ring finger of both hands is shortened so that the last joint of each ring finger can be only slightly flexed. Both clinically and by X-Ray examination, the joints are otherwise normal. This single brief pedigree suggests a dominant trait.

PEDIGREE *B*

In this pedigree the shortening of the flexor digitorum sublimis of the little fingers seems to be a dominant trait, as in Hefner's pedigree, but the family history is not extensive enough to determine whether or not the trait is confined to the women of the family.

PEDIGREE *C*

This pedigree was obtained from a boy, each of whose middle fingers could not be extended beyond a right angle (with the other fingers) at the middle phalangeal joint, due apparently to shortening of the tendon of the flexor digitorum sublimis which is inserted into the sides of the second phalanx about its middle. The joints were all normal and extension of the middle fingers easy and painless up to 90 degrees. His mother was said to have the same anomaly of the little fingers, and a brother of the ring fingers.

PEDIGREE *D*

This is a pedigree of shortening of the flexor digitorum sublimis of the little fingers, such as that reported by Hefner.* He suggested the name streblo-microdactyly (streblos — bent, micros — little, dactylos — finger) but this term would fit equally well the common hereditary anomaly of little fingers that curve outward like a pair of parentheses. Hefner's pedigree showed a simple dominant trait, which is not the case in the pedigree shown above.

Charlotte, N. C. WILLIAM ALLAN, M.D.

*HEFNER, R. A., *Journal of Heredity* 20:395, 1928.

A GENETIC HISTORY OF POLAND-CHINA SWINE

II. "Founders of the Breed," Prominent Individuals, Length of Generation

(Concluded from the April issue)

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IN the first section of this history we traced the origin of the Poland-China breed, and showed how the rather slight inbreeding practiced in the breed as a whole had increased slightly in spite of a drastic reversal in ideals as the "hot blood" was replaced by the "big type." We now propose to consider the individuals which have had an outstanding place in the development of the breed.

Table II shows all ancestors which were found in any one sample in 50 or more of the 800 random lines. The number of lines in which the ancestor was found, divided by the total number of lines (800) in which it might possibly have been found indicates the fraction of the genes in the breed at the date sampled which came through that particular ancestor. Selection for or against the genes which came from a certain ancestor, so far as that was different from selection for or against the descendants of this ancestor, can hardly have made the situation very different from what these fractions indicate, except for a few genes which may have been major objects of intense selection. As estimates of the condition prevailing in the whole breed, the figures in Table II can contain sampling errors from choosing which pedigrees were to be studied, or from the randomness with which each line was traced from the sire and from the dam in the chosen pedigrees. The animals in Table II were selected for that table solely because they were found many times. This *ex post facto* selection probably makes the figures in Table II include more than a fair share of the random errors which tended to make them large and less than a fair share of the random errors in the opposite direction. The standard errors of those numbers provide a basis for discounting these sampling variations. The figures in Table II indicate only the direct relationship of the animal to the breed. If a prominent animal also had many brothers, sisters, aunts, uncles or other collateral relatives which contributed much to the inheritance of the breed, then the total relationship of such an animal to the whole breed will be higher than these indicated.

All the animals shown in Table II were related to each other (many of them closely) in an irregular network of descent. That is shown graphically in Figure 32. The three most recent boars are the only animals in Table II which are omitted from Figure 32. The large number of intervening ancestors made it impractical to include the many tangled lines of their network of descent from this group. In Figure 32 each animal appears but once. Arrows show the lines of descent. Another arrow is used for each additional offspring which connects the ancestor with one of its descendants named in Table II. Animals which are not themselves listed in Table II but which were intermediate between two important animals which were ancestor and descendant, are shown as small circles. Figure 32 shows the kind of tangled network of descent which may connect individuals in one of those loosely defined groups which are sometimes called "families." Many other lines of this network are omitted from Figure 32 either because the common ancestor was not prominent enough to be included in Table II or because the connection is through ancestors registered only in the Ohio or Central herdbooks.

Six animals not shown in Table II were found 40 or more times in at least one sample but not as often as 50 times. Four of these (One Price 18,871, Black Chief 21,567, Chief Tecumseh 3rd 38,-

233, and Mollie King 2d 82,680) are in Figure 32 as circles, the fifth (Longfellow 7th 95,747) is a son of Longfellow 2d 52,999 and is out of a sow who has three grandparents shown in Figure 32. The sixth (The Rainbow 329,731) was born in 1918 and, like The Clansman, Liberator, and The Armistice (but not very closely related to them), is descended in many lines from the group shown in Figure 32. Five more animals were found at least 30 times in at least two samples each but not as often as 40 times in any one sample. Three of those (George Wilkes 14,487, Orange Price 34,825, and B. B. L. 2d 52,834) are represented in Figure 32 as circles, the fourth (Big Bone 137,161) is a son of A Wonder and out of a daughter of Long Wonder, while the fifth (Big Bob 212,613) has Chief Price for a great grandsire and four other boars from Figure 32 as great great grandsires besides tracing to other animals in Figure 32 through many other longer lines. In short, Figure 32 includes nearly all the individuals through which are likely to have come as many as five per cent of the genes of the breed at one time or as many as four per cent of the genes at two different times. The only such animals omitted were four prominent recent boars which were remote descendants and three less recent boars which were immediate descendants in almost all lines from those shown in Figure 32. A large portion of the network of descent of the whole Poland-China breed comes through the 26 animals shown in Figure 32.

Peter Mouw of Orange City, Iowa, was the breeder of seven of the 26 animals listed in Table II and W. A. Jones of Van Meter, Iowa, bred two. If we consider the subsequent owners as well as the breeders of these 26 animals, Peter Mouw was a breeder or at one time owner of 11 while W. A. Jones and also B. L. Gosick of Fairfield, Iowa, each bred or at one time owned four. Peter Mouw, more than any other man, was the leader in producing the "big types" and when those became popular (about 1908 to 1915), the demand for them brought into prominence many animals which were largely of Mouw breeding. The Mouw herd in Poland-Chinas played much the same role as the Cruickshank herd in Shorthorn history, although perhaps subsequent pedigree fashions did not become as extreme in the case of the Poland-Chinas as they did in the case of the "straight Scotch" Shorthorns.

The conflict of opinion between advocates of the "hot blood" and of the "big type" ideals from around 1900 almost to 1920 was extreme. The group of animals closest to Chief Perfection 2d in Figure 32 were much more prominent in the pedigrees of the "hot bloods" than in the pedigrees of the "big types." When the "hot bloods" lost favor soon after 1910, these ancestors lost much of their prominence. This is shown by the way the figures in Table II change from 1910 to 1920. Yet it is worth emphasizing (as Malin[7] has done) that the ancestors most closely identified with the "hot bloods" and those which were prominent in the pedigrees of the "big types" are closely related. Chief Perfection 2d was a grandson of Chief Tecumseh 2d but Grand Chief, whose descendants were so prominent in the pedigrees of the "big types," was a son. The sharp division into contrasting types which existed at one time in this breed seems to have been brought about primarily by a phenotypic selection toward different ideals. As con-

TABLE II. Ancestors Found Often In These Pedigrees

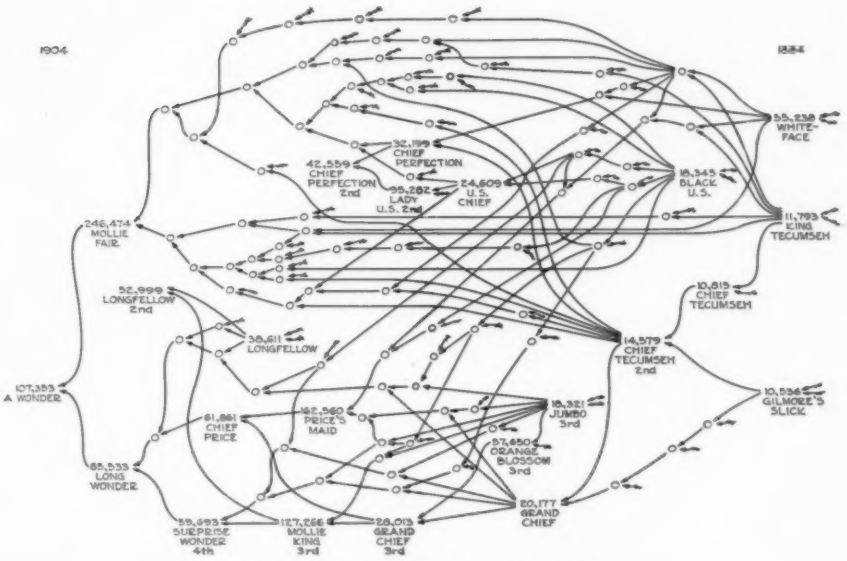
Name	Registration Number	Year Born	Number of times found* among the 500 lines of the sample for: 1900	1910	1920	1929
Sires:						
Chief Tecumseh	10,815	1888	82	82	80	82
King Tecumseh	11,725	1888	60	81	88	70
Chief Tecumseh 2nd	14,979	1892	66	104	119	102
Jumbo 3d	18,321	1899	1	11	86	76
Black U. S.	18,543	1897	40	49	60	58
Grand Chief	20,177	1892	1	16	78	64
U. S. Chief	26,809	1895	29	51	34	5
Grand Chief 3d	29,015	1902	0	19	72	80
Chief Perfection	32,196	1893	15	90	85	36
Longfellow	38,611	1901	1	11	49	37
Chief Perfection 2nd	42,889	1898	22	177	98	37
Longfellow 2nd	52,999	1898	0	9	39	68
Surprise Wonder 4th	59,693	1899	0	15	81	64
Chief Price	61,861	1898	0	88	108	110
Long Wonder	69,333	1903	--	15	81	85
A Wonder	107,355	1904	--	11	87	69
The Clansman	306,869	1918	--	--	16	64
Liberator	358,319	1919	--	--	8	56
The Armistice	459,205	1921	--	--	--	35
Dams:						
Gilmore's Slick	10,856	1894	40	86	77	58
Whiteface	55,830	1898	14	82	15	15
Orange Blossom 3rd	57,452	1891	0	11	46	54
Lady U. S. 2d	93,388	1894	15	75	6	6
Mollie King 2nd	127,806	1896	0	11	86	66
Price's Hold	142,860	1898	0	11	88	64
Mollie Pair	866,674	1903	--	1	84	47

* The standard errors (by the $\sqrt{pq}$ formula) of these numbers are as follows:

Numbers	Standard Error
32 to 44	6
45 to 60	7
61 to 80	8
81 to 100	9
101 to 132	10

TABLE III. Length of Intervals between Generations

Sample born in	Average birth date of foundation animals	Average interval between generations (in years)	Number of generation intervals on which this average is based
1900	1886	2.45	4,113
1910	1886	2.39	6,907
1920	1886	2.49	10,837
1929	1886	2.44	14,287



TWENTY YEARS OF INBREEDING

Figure 32

The tangled network of descent by which the animals of Table II are interrelated. Each animal appears but once. Parent and offspring are connected by an arrow.

cerns the breed as a whole, this is assortive mating which, as Wright[12] showed in 1921, quickly leads to greater diversity of the breed but achieves nearly the limit of its effects in the first two or three generations it is practiced unless (1) the assortive mating is almost perfect — impossible to achieve in practice, of course —or (2) a rigid genetic barrier is erected between the types so that there is no longer any interchange of breeding stock between them.

That there are more boars than sows in Table II is a natural consequence of the average boar's leaving more offspring than the average sow. Yet every animal receives about as many genes from its dam as from its sire (a bit more or a bit less if the sex-linked genes are included) and the characteristics of the population are determined about as much by the dams, considered collectively, as by the sires. A dam who has three or more equally important sons will have more influence on the breed than any one of her sons. That there is more opportunity to improve the breed through choice of males than through choice of females, is true only because a smaller number of the males need be saved for breeding. This gives opportunity for more intense selection among them and more opportunity for chance (in addition to whatever is accomplished by deliberate selection) to cause gene frequency in the chosen males to be different from what it was in the whole population from which they were chosen and therefore to drift a bit from one generation to the next.

Three different animals in Table II were at one time or another the source of more than one-eighth of the genes of the breed, and may fairly be called great grandsires of the breed. Chief Tecumseh 2d was almost equally prominent in the "hot bloods" and the "big types" and his importance to the breed was not altered by the change in ideals. He contributed 13 to 15 per cent of the genes in each of the three recent samples, and more than ten per cent in the 1900 sample, only ten years after he was born. Chief Perfection 2d contributed nearly 17

per cent of the genes in the breed of 1910 but the "hot bloods" were then enjoying their greatest popularity. After that popularity waned, he fell back to a minor position. Chief Price, who is sometimes called the "Father of the Big Types," contributed around 12 to 13 per cent of the genes of the breed in both the 1920 and 1929 samples.

These figures emphasize the important fact that one individual can become prominent in a population as large as this entire breed. In the period from around 1888 to around 1898 (when most of the animals shown in Table II were born) this association was registering something like two or three thousand boars per year and more than twice that many sows. How could one animal exert on a population as large as this as much influence as Table II shows that some did? The opportunities which individual animals have for contributing to the inheritance of the future breed are by no means equal. Certain animals, partly by their own merit and partly because of other incidents, are used in herds which (again partly as a matter of chance or economics or salesmanship and not solely on their biological superiority) become famous as sources of breeding stock and manage to continue this prominence for considerable time. These animals have many times the average opportunity to contribute to the inheritance of future generations. Many registered animals go to grade herds. Many others are used in herds which sell breeding stock only to grade herds. Their inheritance is diffused into the population of grade animals which produce the market stock and is lost from the inheritance of the future breed. Thus the number of sires which contribute to the maintenance of the breed is considerably reduced.

As concerns the part played by chance in altering gene frequencies in pure breeds, the effective population number is far smaller (in this breed less than one per cent as large) as the census number of those which reach breeding age. For example, the three foundation animals which were found most often in the 1929 sample were the two boars, Jumbo 3rd and King Tecumseh, and the sow, Gilmore's Slick. Together these three contributed 25 per cent of the genes of the 1929 sample, 26 per cent to the 1920 sample and 15 per cent to the 1910 sample. Since each animal doubtless contains many hundreds and perhaps several thousands of pairs of genes, it can easily have happened that these three would all have been like each other but different from the average of the breed in respect to some pairs of genes. If so, whatever choices or chances determined that these three animals should be used so extensively can have shifted the frequency of each such gene far to one side of what it would have been if it had been made rarer or more abundant solely because of selection against or for its own average effects. This would be especially true of genes which had minor effects. Genes which had major effects would be more closely guided by selection, but selection could be intense for each gene only if few genes were concerned. The ten foundation animals found most often in the 1929 sample are at the ends of 45 per cent of the lines. The same ten are at the end of 46 per cent of the lines in the 1920 sample and 30 per cent in the 1910 sample. This gave opportunity for gene frequency to wander by chance farther than it could have done if no one animal had contributed a large share of the genes.

When considering problems of evolution we would like to know how far this situation finds its parallel in populations in nature. It is conceivable that this extreme contrast between the census number and the effective number in populations of farm animals is brought about primarily by conditions peculiar to domestication. It may be that economic conditions, potency of advertising, prominence given by show ring awards, etc., leave room for only one or two or at most a few animals to be famous at a given time. The desire of all breeders to have sons or grandsons or other close relatives of the currently famous animals at the head of their herds would lead to a rapid diffusion of the genes of those few through the breed and to a corresponding diminution of the chances that any genes carried only by the less popu-

lar animals would continue to exist in that breed. Does anything corresponding closely to this exist in natural populations?

How Many Years Per Generation?

By noting the birth dates of the animals at the end of each ancestral line and counting the number of generations intervening between that foundation animal and its descendant, we can find the average length of time which elapsed from one generation to the next. Those averages and the amounts of data on which they are based are shown in Table III. Most sows and boars have their first litters born when they are about one year old, but enough sows and boars are kept in use until they are three or four or more years old that *the average age* of the parents when the pigs are born is raised from the physiological minimum of about one year to the two and one-half years actually found. Since these figures come only from the pedigrees of registered animals, they may be somewhat longer than is typical of general practice in swine production. If breeders prefer to save breeding stock only from proved sires and dams, they doubtless register a larger share of the pigs which are from older sows and boars than of those from young sows and boars. The average intervals in Table III may be a little larger than the average ages of all purebred sows and boars when their pigs are born.

If a breeder wishes to accomplish in the minimum time a plan which requires a certain number of generations, it would be possible to have the generation intervals as short as one year. In an inbreeding experiment, for example, if one wished to attain a certain intensity of inbreeding as quickly as possible, he could accomplish ten generations of inbreeding in ten years by breeding only from gilts and young boars. The same amount of inbreeding would require some 25 years if the experimenter followed the customary practices followed by most breeders with respect to age of boars and sows. Saving time by using only gilts and young boars would cost him much of his opportunity to practice selection. He could not keep for second or third crops of pigs those young sows and boars whose offspring prove them best or those sows which showed unusual performance as nurses. In some breeding plans, therefore, the wisest policy with respect to age will be some compromise between the maximum use of selection, which could be had by using the better ones among the sows and boars as long as they seem to be better than the average young boars or gilts, and the maximum economy of time in reaching a given degree of linebreeding, which could be had by producing a generation each year.

Discussion

Malin's is the only other study[7] of the Poland-China breed from a viewpoint similar to this. Malin started with a sample of prize-winners from which he located the most prominent sires, grandsires, and great grandsires of prize-winners. He then analyzed the pedigrees of those prominent sires to learn what ancestors had been most important and what breeding systems had been used in producing them. He interprets his findings as showing more inbreeding and also more distinct splitting into families than we were able to find, but that comes mainly from his use of "percentage of blood" which is fairly accurate for expressing relationship between ancestor and descendant but makes the intensity of inbreeding seem higher than it really is. Otherwise the results are generally similar, including the part about the ancestors which played a prominent part in the "hot bloods" and those which played a prominent part in the foundation of the "big types" and their common origin and close relationship to each other. There are some minor differences as, for example, his statement (page 199) that the average of Chief Price, Longfellow, and Jumbo 3d blood in the breed has been declining since 1910. Our findings contradict that and suggest that the part which these boars play in the pedigrees of the breed may be almost stabilized so that it cannot any longer be raised or lowered very much. Also Malin says that present-day (1923) Poland-Chinas show a negligible quantity of the blood of the boar, Expansion 57,691, but we

found him seven times in the 1910 sample, 21 times in 1920 and 25 times in 1929. While that is distinctly less frequently than many other animals, yet it indicates that something like three per cent of the genes of the present breed come through him.

Rottensten's study[8*] of the Danish Landrace included only one sample, — from Volume 21 of the herdbook and averaging 1930 for birth date. He found that the inbreeding coefficient had risen to 6.9 ± .7 per cent in the period from 1897 to 1930. This is about one half per cent per generation. This was even less than was expected from the average *inter se* relationship coefficient (16 ± 1.7 per cent), thus indicating steady efforts by Danish breeders to avoid even mild inbreeding. The highest relationship of any one animal to the whole breed was twelve per cent. The average interval between generations was 2.2 years. These results are surprisingly similar to our findings, in view of the difference in registration customs, emphasis on progeny testing, etc., in the two countries.

What would we have found if we could have carried this study back to the earliest foundation period, that is to 1845 or thereabouts, when the last introduction of distinctly outside blood is reputed to have taken place? That would be about 16 generations farther back than the base date of the present study. With respect to the real formation of the Poland-China breed our study perhaps may show only as incomplete a picture as a study of the Shorthorn breed if pedigree lines could not be traced farther back than 1850.

Random errors in the herdbook or in the computations would have the general bias of making these figures for the intensity of inbreeding or for the importance of individual ancestors lower than they should be, since the alternatives to which a mistake in one digit might change an already prominent number are many. Errors arising from deliberate fraud in registration would tend in the opposite direction. Naturally there is no good way to estimate how many errors of that kind occur, but a man who makes one such fraudulent registration is apt to keep on making them until eventually he is exposed. When such a case is exposed, breeders tend to avoid or discard all animals ever registered by that man. The breed thus tends to purge itself of pedigrees containing false entries. While we would not maintain that this process is complete, we do think that the undetected errors from this source are so few that they cannot have had much effect on our findings.

Summary

1. The average rate of inbreeding in the Poland-China breed of swine from 1886 to 1929 was enough to lose about six-tenths of one per cent of the existing heterozygosis per generation. Samples of the breed from 1900 to 1929 indicate little change in the average inbreeding rate, even though the breeding policy underwent wide changes in that time.

2. There was only a faint tendency for distinct families to be formed and kept free from interbreeding with each other.

3. Three ancestors each contributed more than twelve per cent of the genes of the breed, unless selection against those genes has been extreme and effective. One of these was an ancestor of the other two. The ten foundation animals found most often in the 1929 sample contributed 45 per cent of the genes of the breed then, 46 per cent in 1920 and 30 per cent in 1910. More than half of this came through three of them. This situation could have permitted gene frequency in the breed to drift far without any selection or even against moderate selection.

4. The principal plan of breeding seems to have been selection and extensive use of the sons and daughters and grandsons and granddaughters of the currently most famous sires and dams in the breed, with the spotlight of that current fame sometimes shifting from one animal to another not very closely related to the reigning favorite which it displaces. Most of the inbreeding has been incidental to this.

5. The average interval from one generation to the next is about two and one-half years.

*Literature references were published in the April issue.

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